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Original Articles and Clinical Cases.

SPINAL CORD CHANGES IN CASES OF CEREBRAL TUMOUR.

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(1) Introduction and general statement of the changes found in the spinal cord in cases of cerebral tumour.
(2) Synopsis of previous observations on this subject.
(3) Suggested explanations of the changes found.
(4) Analysis of the cases.
  (*a*) Character of the degeneration.
  (*b*) Nature and situation of the tumour.
(5) Discussion of the cause of the spinal cord degeneration.
(6) The effect of operation; the condition of the knee-jerk and arm-jerks, and the presence of optic neuritis.
(7) Conclusions.

Appendix.
  Table of cases.
  Details of clinical history and pathological results.

I.

WHILE examining the spinal cords from cases that died of cerebral tumour, the fact that a considerable amount of degeneration was present in the posterior columns became apparent, and in order to determine what relation these

changes bore to the symptoms present during life, and also to ascertain how frequently and to what extent these changes occurred, as many cases of cerebral tumour as could be obtained were examined, and, having lately had the opportunity of investigating some twenty-nine cases, we venture to bring the results under your notice. For the details of the clinical side of the cases, one of us (J. S. C.), and for the pathological investigation of the cases, the other (F. E. B.) is alone responsible, but for the views expressed we are jointly responsible. For permission to make use of these cases we would here acknowledge our indebtedness to Dr. Hughlings Jackson, Dr. Buzzard, Dr. Bastian, Sir William Gowers, Dr. Ferrier, Dr. Ormerod, Dr. Beevor, Dr. Tooth, Dr. Taylor, Dr. Colman, and Dr. Risien Russell, of the National Hospital, and to Dr. Lees, of the Children's Hospital.

II.

Not a few authors have already written on this subject, and it will, therefore, be most convenient to give a short summary of their observations before we proceed to deal with our own cases.

Historical.

Degeneration of the posterior columns in cases of cerebral tumour has been noticed and recorded by several observers. C. Mayer, in 1894, in a paper, "Ueber anatomische Ruckenmarks befund in Fällen von Hirntumoren," was the first to recognise that the condition was associated with certain clinical phenomena, and to discuss its pathology.

In the above paper he refers to a paper published by Wollenberg, who described, in the case of a tumour of the left cerebellar hemisphere, a typical posterior degeneration extending from the lumbar to the cervical region, and affecting also the ascending roots of the fifth nerve and the posterior nerve-roots. The knee-jerks were absent, and the condition was regarded by Wollenberg as one of commencing tabes.

Two cases are described by Mayer, with loss of knee-jerk and early degeneration of the posterior columns.

Case 1 was that of a woman, aged 33, with aphasia and right hemiplegia, double optic neuritis, absent knee-jerks, but with active Achilles jerks. At the autopsy a tumour was found in the third left frontal region and operculum, and, on microscopical examination, degeneration was found in the posterior columns, but little marked in the sacral and lower lumbar region, more marked in the upper lumbar, dorsal, and cervical regions, and most marked in the column of Burdach in the last-named region. Lissauer's tract was unaffected, and no degeneration could be found in the extra-medullary portion of the posterior roots. Changes were noted in the cells of the anterior horns, but Clarke's column appeared quite normal.

In the second case, a man, aged 46, in whom symptoms had lasted for one year, and in whom the knee-jerks were absent, a tumour was found growing from the anterior extremity of the falx, and pressing on the frontal lobes. On examination of the cord, degeneration was found in the posterior columns, and especially in Burdach's column, and although some degeneration existed in the dorsal region, it was most marked at the level of the lower cervical roots. No degeneration could be found in the extra-medullary roots, and Clarke's column appeared to be normal.

That the condition is not one of early tabes is shown by the fact that the degeneration was more marked in the cervical than in the lumbar region of the cord, and affected the columns of Burdach to a greater extent than the column of Goll, and Lissauer's tract was unaffected.

That the condition cannot have been due to an affection of the spinal meninges was proved by the complete absence of any pathological change in these membranes.

In discussing the pathology, the author of this paper calls attention to the fact that in certain diseases the posterior columns are very liable to be affected, viz., ergotism, pernicious anæmia, lead poisoning, leprosy, alcoholism, Addison's disease, and like conditions; and he suggests that the degeneration above described may be due to some toxin in the altered cerebro-spinal fluid, which has been supposed to give rise to optic neuritis. Against such a

theory the fact that the extra-medullary spinal roots, which are bathed with this fluid, escape, is brought forward. The question of the condition being due to increased intra-cranial pressure is then discussed, and, lastly, the condition of venous stasis produced by such an increase of pressure, the latter condition being thought the most probable origin of this degeneration.

In 1894, Campbell read before the British Medical Association, at Bristol, an account of two cases of softening of the cerebellum due to vascular change, associated with degeneration of the posterior columns.

In Case 1, a woman, aged 62, there had been paralysis of the left arm and leg for two years before death, and there was old softening and destruction of the greater part of the left cerebellar hemisphere, with degeneration in the left middle, inferior peduncles, the direct cerebellar tract, the ascending antero-lateral tract, and the posterior external columns of the left side; alteration in the nerve-cells in the region of the anterior cornu of the cervical region, and also degeneration in the left lateral pyramid. Clarke's column also appeared small.

In Case 2, a man, aged 73, with weakness of the left arm and leg, softening was present on the upper surface of the left cerebellar hemisphere, and degeneration was present in the left superior, middle, and inferior cerebellar peduncles; in the nucleus and funiculus cuneatus and in the posterior external columns of the cord. At the level of the sixth cervical root the degeneration occupied the outer half of Burdach's column. No degeneration could be traced below the level of the sixth dorsal root.

The author regards the degeneration of the direct cerebellar tract as a secondary atrophy, and not as a true degeneration; but the posterior degeneration he is inclined to regard as due to this part of the postero-external columns, being a direct descending cerebellar tract connecting the cerebellum with the lateral group of the anterior cornual cells of the lower cervical region of the same side. In a private communication, September, 1899, he states he is inclined to believe that this posterior degeneration is also a retrograde change, and not a true degeneration.

In 1895, Dinkler recorded a case of hydrocephalus and tumour of the right parietal lobe, with absent reflexes, associated with degeneration of the posterior columns, most marked in the cervical region and in the column of Burdach, and present also in the lumbar region of the cord. There was degeneration of the posterior roots. With regard to the pathology of this condition, after mentioning, he at once negatived, the possibility of the condition being due to involvement of the roots by growth secondary to that present in the brain, and suggested two other possible explanations:—(1) That the tumour produced some toxic substance; (2) that the condition was due to cachexia, since Oppenheimer and Siemerling have shown that degeneration of peripheral nerves may occur in cases dying of carcinoma, and the author inclined to this explanation of the condition.

In 1896, Pick described five cases:—

(1) A tumour in the posterior and median portion of the left cerebral hemisphere, with degeneration in the pyramid and in the posterior columns and in the posterior roots.

(2) A tumour in the left cerebral hemisphere, with degeneration most marked in the posterior columns in the lower cervical region, but also present in the lumbar cord.

(3) An osteo-sarcoma of the skull in a child, with degeneration most marked in Burdach's column, the posterior roots being unaffected.

(4) A glioma of both cerebral hemispheres, with no degeneration.

(5) A diffuse cerebral sclerosis, with degeneration of the posterior columns, most marked in the lower cervical region.

The author concluded that the condition was due to increased intra-cranial pressure, but why certain areas of the cord and roots were more affected than others he left for further observations to explain.

In 1896, Besold, in the case of two sisters, described degeneration of the posterior columns of the cord, most marked in Burdach's column and in the cervical region, associated with tumours in region of the third ventricle and optic thalamus. Degeneration was also present in

Gower's tract. The author attributed the condition to anæmia.

In 1897 Ursin described three cases :—

(1) A tumour in the left cerebral hemisphere with degeneration in the postero-external columns, leaving the septo-marginal, the cornu-commissural zone, and Lissauer's tract unaffected. The extra-medullary portion of the posterior roots were affected, and some alteration in the cells of Clarke's column was present in the dorsal region. The degeneration was most marked in Burdach's column, and was not present above the posterior column nuclei.

(2) Two tumours, one in the left occipital lobe and another in the right frontal lobe. The cord showed degeneration in the posterior columns most marked in Burdach's column. The posterior roots, Lissauer's tract, and Clarke's column showed no change. There was degeneration in the lateral columns.

(3) A tumour in the left optic thalamus, with degeneration in both pyramidal tracts, diffuse degeneration in the posterior columns, and also in the intra-medullary portion of the hypoglossal nerve of the right side. No degeneration was present in the extra-medullary roots or in Lissauer's tract.

He considered that the condition was not due to increased intra-cranial pressure, but to an intoxication and a failure of nutrition.

In 1897 Hoche described three cases :—

(1) A tumour in the right cerebral hemisphere with degeneration in the left pyramid and in the posterior columns. The extra-medullary roots were unaffected, and the change was more marked in the cervical than in the dorsal or lumbar region.

(2) A tumour in the left lateral lobe of the cerebellum, with degeneration in the posterior columns, more marked in the cervical and lumbar regions than in the dorsal. The extra-spinal roots were unaffected.

(3) A tumour growing from the dura mater, and pressing on the left cerebral hemisphere. The spinal cord showed no degeneration.

The author concluded that neither the age of the patient nor the situation, nor the histological characters of the growth, were in causal relationship with the degeneration of the posterior columns, but that the condition was dependent upon increase of intra-cranial pressure.

In two of the cases above reported there was optic neuritis and degeneration of the posterior roots, while in the third there was no optic neuritis and no degeneration of the posterior roots.

In 1899 Bikeles described posterior degeneration occurring in a patient who died twenty days after being shot in the right frontal region. The injury gave rise to left hemiplegia. There was degeneration in the right pyramid and in the column of Goll from the mid-dorsal to the cervical region. This degeneration was most marked on the side of the affected pyramid. There was also degeneration of the first cervical root on the right side.

The suggested explanation of the condition was that the disturbance caused by the bullet wound was conveyed to the spinal cord through the cerebro-spinal fluid and affected the most vulnerable portions, viz., the posterior columns, although the effect of toxin could not be excluded since suppuration and encephalitis were present in the region of the injury. With regard to the first cervical root which showed degeneration, not only in its intra-spinal course, but also in its extra-spinal course, it was suggested that the sudden movement of the cerebro-spinal fluid owing to the entrance of the shot would be especially liable to injure this root on account of its close proximity to the foramen magnum.

Digest.

In the above papers there are fifteen cases of cerebral tumour showing degeneration in the posterior columns and two cases with negative results so far as the cord is concerned. In addition to these there are two cases published by Campbell with degeneration in the posterior columns in which there was no tumour, but thrombosis of some cerebellar vessel. In twelve of the above cases, at any rate, the

degeneration was most marked in the lower cervical region, and in only four of these was there degeneration in the extra-spinal posterior roots.

III.

It will be gathered from the above digest that various suggestions have been brought forward to account for this posterior degeneration, and of these the following may be at once rejected:—

(1) That the condition is one of early tabes.

(2) That the condition is due to involvment of the posterior roots by secondary deposits from growth in the cranium.

(3) That the condition is secondary to some spinal meningitis or thickening of the membranes.

(4) That the condition is due to a descending degeneration in the posterior columns. (Under this heading retrograde degeneration is not included, as this will be discussed later on.)

Passing now to the consideration of the various conditions which may reasonably account for this spinal change, one may discuss them under the following heads:—

(1) A toxic agent. (*a*) Produced by the tumour itself. (*b*) Secondary to the anæmia and cachexia resulting from the tumour.

(2) Increased pressure in the cerebro-spinal canal.

(3) Venous stasis, anæmia, thrombosis in the spinal vessels secondary to the increased pressure.

(4) Retrograde degeneration.

(1) (*a*) *A toxic agent produced by the tumour.*—Assuming that it is possible for a malignant growth to produce a toxin either by its own activity or by the destructive change in the cerebral substance in which it grows,[1] is it possible that such a poison would exert a selective action on certain portions of the nervous system? (for instance, the posterior

[1] MOTT AND HALLIBURTON, *Philosophical Transactions*, vol. cxci., p. 211-267. Mott and Halliburton have shown that the body "choline," formed from the destruction of lecithin, is present in the cerebro-spinal fluid of patient suffering from general paralysis of the insane, and that injected into animal it produces certain toxic effects.

nerve roots). In reply to such a question the state of things met with in tabes, pellagra, and ergotism, besides other conditions, might be cited as instances where a poison exerts such a selective action. On the other hand, the selective action in the present case seems almost more limited than in the conditions above named, for almost all observers are agreed that the degeneration affects the lower cervical region to a greater extent than any other portion of the cord, and the same is borne out in the present series of cases, though it is by no means invariable, for in two cases (Nos. 19 and 27) the degeneration was as marked in the lumbar region of the cord as in the cervical.

That posterior degeneration is more marked in cases where the tumour is of rapid growth and invades the cerebral substance is certainly born out by the present series of cases, but there are other conditions accompanying such rapid growth which may exert a harmful influence on the posterior roots, and the effect can hardly be attributed to the action of a toxin.

(*b*) *A toxic agent secondary to the anæmia and cachexia produced by the growth.*—It is hardly possible to maintain such a view, for *it is well known that* patients suffering with cerebral tumours rarely emaciate to any marked extent, and are often well nourished. In 140 consecutive cases of intra-cranial tumour examined by one of us (J. S. C.) at the National Hospital, general wasting was observed in seven cases only, and in each case a complication was present to account for such wasting, *i.e.*, tubercular meningitis, once; tubercular peritonitis, once; carcinoma of lung, once; prolonged stupor with the attendant difficulties of administering nourishment, four times.

The marked hemiplegic and general wasting, not infrequently observed after the removal of intra-cranial growths by operative measures, is not considered among these cases.

In six cases where the symptoms were of long duration (upwards of eighteen months), and where the growth was of large size and situated in the cerebral hemispheres, the patients were very well nourished at the time of death.

In the series of cases here recorded wasting was a marked

feature in four cases only, Nos. 5, 12, 18, and 21, and these cases were well nourished until operative measures for the removal of the growth had been resorted to. In the cases, Nos. 18 and 21, where emaciation was extreme, no degeneration of the posterior columns was present.

It should, however, be borne in mind that Oppenheimer and Siemerling have shown that changes are sometimes found in the peripheral nerves in cases of carcinoma. That the condition cannot depend on simple marasmus is shown by the fact that in three cases of extreme wasting due to tubercle examined by Mayer no change could be found, nor can it be due to anæmia alone, for I have examined cords in extreme anæmic conditions with a negative result.

(2) *That the condition is due to increased pressure in the cerebro-spinal canal* is a view that would seem to be the most reasonable explanation, and even if it is not the sole cause of the condition, it is most certainly an important factor in the ætiology of the condition. There are, however, various arguments against such a view.

Firstly, it is urged that if the condition is due to increased pressure, then the degeneration should be most marked in the lumbar region where the fluid pressure is greatest. On the other hand, the point at which the degeneration is most marked is the cervical region, an objection, however, that does not carry much weight in that many patients who present these changes in the cord are unable to maintain the erect position, and, therefore, in them the pressure is not greater in the lumbar than in the cervical region.

Secondly, there are cases in which there is no increase in the intra-cranial pressure, and yet degeneration of the posterior columns has been present. Moreover, the cases reported by Dr. Campbell, of Rainhill, might be cited in this connection.

Thirdly, the degeneration is stated to be often asymmetrical, whereas if it had been due to pressure one would have expected it to have been symmetrical. In this relation, however, it must be pointed out that the asymmetry of the lesion is very slight as a rule, so that little can be based on this point.

Fourthly, the degeneration is often limited to the cord and the posterior roots are unaffected, whereas the roots are more exposed to pressure than is the cord.

Such are the arguments against the pressure theory, but there is no doubt that the degeneration is present in its most marked extent in those cases in which the rise of intracranial pressure has been rapid. In the chronic cases where there is marked hydrocephalus the degeneration is often slight or absent.

(3) *Venous stasis, anæmia, or thrombosis in the spinal vessels secondary to the increased pressure.*—Direct evidence on this point is wanting, for in all the cases examined there is no evidence of thrombosis of the smaller vessels; sometimes they are full and engorged, at other times empty. However, the change in the cord is never a localised patch of softening or myelitis such as one might expect to find if there were actual thrombosis of some of the smaller vessels, nor is there anything to suggest that the change is due to hæmorrhage. There is no evidence pointing to the posterior root ganglia being the seat of the lesion; indeed, such evidence as there is is directly opposed to this, for in those cases in which the ganglia have been examined they have been found to be normal. The posterior roots are often unaffected, and even when affected the degeneration is always less than in the posterior columns of the cord. The affection of the direct cerebellar tract is uncommon, and does not necessarily occur in cases of cerebellar tumour. In one case (No. 1) in which it was most marked it was symmetrical, and only extended down to a point between the first and second cervical. There were a few degenerate fibres below this level, but they could not account for the marked degeneration above this level. There was no evidence of myelitis in this region, but that there must have been some pressure at this level, viz., first and second cervical, is borne out by these two facts, viz., that there was marked degeneration in the direct cerebellar tracts above that level, and there was an increase in the degeneration in the pyramidal tracts below that level. Such a condition is not common, and occurred in only two other cases (Nos. 5 and 20), and in the

former of these the degeneration in the posterior columns only became marked above the level of the second cervical.

(4) *Retrograde degeneration.*—This view was advanced by Dr. Campbell in his most interesting report of cases of softening of the cerebellum, with degeneration in the posterior columns and the direct cerebellar tract, but although his arguments are applicable in the cases he brings forward, yet such an explanation cannot account for the condition met with in the present series of cases, except so far as the extra-spinal posterior roots are concerned. This point will be further discussed in the latter portion of this paper.

IV.

Twenty-nine cases have been examined, and the accompanying table indicates their situation and the amount of degeneration that occurred in the posterior columns. For the sake of classification the degeneration has been placed under the three heads, "Marked," "Slight," and "None."

TABLE I.

	No. of cases.	No degeneration.	Slight degeneration.	Marked degeneration.
Frontal	6	2	2	2
Parietal	2	1	1	0
Temporo-sphenoidal ..	7	1	2	4
Cerebellar	9	3	2	4
Basal ganglia and pons ..	5	2	2	1
	29	9	9	11

From the analysis shown in Table I. it would appear that degeneration is rather more liable to occur with tumours in the temporo-sphenoidal region than when situated elsewhere, and also that a tumour situated behind the parietal region is more liable to give rise to degeneration than one more anteriorly situated.

(*a*) *Character of Degeneration.*

The degeneration is of such a character that though readily shown by the Marchi method, is not well-marked by the Weigert-Pal method. The degeneration affects the

posterior columns of the cord, and in all cases, except No. 27, is more marked in the cervical than in other regions. It therefore follows that the postero-external column (Burdach) is more affected than the postero-internal (Goll). The degeneration is of root origin, for in many cases it can be seen limited to a region occupied by the ascending fibres of a root, and the endogenous tracts tend to escape. It cannot be said, however, that these tracts contain no degenerate fibres, but fewer than the tracts of exogenous origin; and although the endogenous tracts are, for the most part, composed of fibres derived from within the cord, yet few will deny that they contain also fibres of exogenous origin.

In one case, No. 6, the degeneration was limited to one root, viz., the fifth lumbar, and this degeneration was so marked that it might be considered fortuitous; and in another case, No. 15, it was limited to two roots, the sixth and seventh cervical. In the cases 10, 11, 19, 27, degeneration was well marked in the whole of the posterior columns of the cord, from the cervical to the lumbar region, but in the remaining cases the condition, although not limited to the cervical region, showed comparatively little or no degeneration in the dorsal and lumbar region.

The degeneration could be traced up to the posterior column nuclei, and as the column of Burdach was most affected in the cervical region, so the degeneration was more marked in the funiculus cuneatus than in the funiculus gracilis.

Lissauer's tract.—A few degenerate fibres were present in this tract in two cases, Nos. 10 and 11, but in neither was this marked, and in the remaining cases the tract appeared perfectly normal.

The fillet.—Slight degeneration was present in five cases, Nos. 5, 11, 12, 26, and 27, but in none was it marked nor in any way proportional to the amount of degeneration in the posterior columns.

The direct cerebellar tract.—There was degeneration in this tract in three cases; in all three it was symmetrical, and in case No. 1 it was only present above the level of the second cervical root; in cases Nos. 12 and 20, as no exam-

ination of the cord was allowed, it was impossible to say from what point it extended. It has been shown by Dr. Leonard Hill ("Allbutt's System of Medicine," vol. vii.), that when the intra-cranial pressure is rapidly increased, the cerebellum and the bulb descend and completely block the foramen magnum, and, from the altered position of the parts, pressure must be brought to bear on the upper region of the cervical cord, and this gives a ready explanation for the degeneration in the direct cerebellar tract.

Posterior roots.—Degeneration of the posterior roots was present in five cases, and in one other to a slight extent; it was always considerably less than the degeneration in the posterior columns.

Posterior root ganglia.—In a few cases these have been examined, but with negative results.

Cranial nerves.—In case No. 1 the intra-medullary portion of the twelfth nerve on one side was found to be degenerate; the cause of this degeneration could not be found, but as there was a localised patch of softening in the upper portion of the pons, a similar patch may have occurred near the twelfth nucleus, and thus have given rise to this degeneration.

In case No. 12 there was degeneration of the seventh nerve on one side; the origin of this also was not found.

(*b*) *Nature of Growth.*

The growth varied greatly, not only in external but also in microscopical appearances, and the following table gives the number in each group:—

TABLE II.

Nature of growths.

Tuberculous	2
Gummatous	1
Glioma	5
Psammoma	1
Cyst of choroid plexus	1
Sarcoma (under this head are included fibro-alveolar, glio-, and irregular-celled sarcomata as well as endothelioma)	18
Not examined	1

V.

The cause of the spinal cord degeneration may be conveniently discussed under the following heads.

(1) Does the condition depend on the situation of the tumour?

(2) Does the condition depend on the nature of the tumour?

(3) Does the condition depend on the increase of intracranial pressure and the rapidity with which such increase occurs, or is it a combined effect of the increased intracranial pressure with dilatation of the ventricles, or is it dependent on the latter factor alone?

(1) *Does the condition depend on the situation of the tumour?*

In answer to this question, if one takes the cases in which the posterior degeneration is most marked, viz., 1, 10, 11, 12, 19, 27, one finds that the first is a frontal tumour, the next three are temporo-sphenoidal, the fifth a cerebellar tumour, and the sixth a tumour of the corpora quadrigemina; that is to say, degeneration may occur in whatever portion of the brain the tumour is situated, but that growths in the temporo-sphenoidal region are more frequently accompanied by marked degeneration than tumours in other situations, and the same is borne out if one takes all the cases in which posterior degeneration occurs, as shown in the following table:—

TABLE III.

Frontal	4
Parietal	1
Temporo-sphenoidal	6
Cerebellar	5
Basal ganglia	3

It is, however, obvious from consideration of the above figures that the situation of the tumour alone can have little influence on the occurrence of this posterior degeneration.

(2) *Does the condition depend on the nature of the tumour?*

In considering this question, if one again takes the cases in which degeneration is most marked, viz., 1, 10,

11, 12, 19, 27, one finds No. 1 is a glio-sarcoma, No. 10 is a glioma, No. 11 is a spindle and round-celled sarcoma, No. 12 is an irregular-celled sarcoma, No. 19 is a glioma, No. 27 is a round-celled sarcoma; that is to say, all are comparatively rapidly-growing tumours. On the other hand, if one takes the cases with no degeneration, one finds No. 2 is a sarcoma, No. 3 is a gumma, No. 7 nature uncertain, No. 14 is a spindle-celled sarcoma, No. 16 is a glioma, No. 18 is a tuberculous growth, No. 21 is a fibro-sarcoma, No. 28 is a glioma, No. 29 is a cyst of the choroid plexus; that is to say, a mixed variety of tumours, the majority of which were of chronic nature, but three, at any rate, were of rapid growth. Therefore, it would seem that though the nature of the tumour may be a factor owing to its rapid increase in size, it cannot be considered to play any part with regard to the production of posterior degeneration so far as any toxin which it is supposed to form is concerned.

(3) *Does the condition depend on the increase of intra-cranial pressure and the rapidity with which such increase occurs, or is it a combined effect of the increased intra-cranial pressure with dilatation of the ventricles, or is it dependent on the latter factor alone?*

Nearly all the cases showed increase of intra-cranial pressure either at the time of death or previous to operation. In two cases of long standing (16 and 21) there was no evidence of increased intra-cranial pressure, the lateral ventricles being, however, in both cases very considerably dilated but the cord in these cases showed no change. This would seem to suggest that dilatation of the ventricles alone does not give rise to posterior degeneration.

Taking then the case in which the posterior degeneration was most marked, viz., No. 27, it was found that a large rapidly-growing tumour (nine weeks) existed in the optic thalamus and corpora quadrigemina, causing obstruction to the Sylvian aqueduct and marked dilatation of the ventricles. The degeneration extended the whole length of the spinal cord and affected both the columns of Burdach and Goll. Such a case would seem to point to the condition

being due to the rapid increase in intra-cranial pressure, together with dilatation of the ventricles. This is not, however borne out on examination of the other cases in which the degeneration is most marked, for in the cases 11 and 19 there was no distention of the ventricles. Increased intra-cranial pressure was, however, present, and in one of the cases was limited to the subtentorial region. The one common factor in nearly all the cases seems to be increased intra-cranial pressure, but even when this is present and has been of rapid onset, cases occurred (Nos. 2, 14, 28, 29) in which no spinal degeneration was present. These exceptions, however, admit of an explanation. No. 2 was a frontal tumour, and posterior degeneration seems to occur less frequently with tumours in this situation, and in Nos. 28 and 29 the pressure symptoms had existed a comparatively short time.

The evidence, as gathered from the above series of cases, seems to point to the rapid increase of intra-cranial pressure, especially when associated with dilatation of the ventricles as the cause of the condition. The question then arises: How does such intra-cranial pressure account for degeneration in the posterior columns of the cord? In an article by Dr. Leonard Hill on the experimental pathology of cerebral circulation, he shows that the intra-cranial pressure does not exceed the normal cerebral venous pressure, and makes the following statement:—"Since at any pressure above cerebral venous pressure the cerebro-spinal fluid rapidly leaks away from the cranial cavity, this fluid can be entirely neglected in dealing with the pathology of compression. It is absolutely erroneous to teach that increased cerebral pressure is transmitted to all parts of the cranio-vertebral cavity by the cerebro-spinal fluid. It is equally erroneous to teach that a foreign body within the cranium exerts pressure."

Such a statement as the above makes it extremely difficult to account for posterior degeneration on the increased pressure theory. On the other hand, when one sees the distended ventricles, the dilated posterior cistern and the distended arachnoid space in the spinal cord, one cannot but think that the increased fluid pressure within the ven-

tricles is communicated to the spinal canal. In order to obtain further evidence on this point the spinal canal and the theca were carefully opened so as not to injure the arachnoid; fluid was injected into the ventricles under a pressure of a column of water 2 ft. high, and as the arachnoid became distended it was seen that the spinal roots which, under normal conditions, lie close to the cord and pass in a nearly straight line from their point of exit on the dorsal side of the cord to their point of passage through the theca, were carried away from the cord owing to their attachment to the arachnoid, and formed a bow instead of a straight line, the curve being most acute where the roots passed into the spinal cord. The anterior roots being less attached to the arachnoid membrane than the posterior would be less liable to suffer when the sub-arachnoid space is distended. If the pressure between the dura mater and the arachnoid be the same as that in the sub-arachnoid space, then no stretching of the roots will take place; and this fact may account for the absence of degeneration in certain cases of marked intra-cranial pressure. It is therefore probable that the point at which the root is most likely to suffer is that at which it enters the spinal cord. And it has been shown that at this point it becomes constricted, and the medullation of the fibres becomes thinner and often seems almost to disappear altogether leaving the naked axis-cylinder; traction, therefore, either in the normal direction of the root, or still more in some altered direction, would be extremely liable to injure it at this point.

Now, the fact that the posterior columns of the cord are often affected without the roots showing any or very slight change, is proved in cases Nos. 11, 13, 27, in which the cord is affected, and little or no change could be found in the roots; and in other cases where degeneration is present in the roots it is slight in comparison to the amount of change in the posterior columns of the spinal cord. The explanation of such a condition is clear, namely, that degeneration occurs in the portion of the neuron distal to the injury, *i.e.*, that in the posterior columns, while only a slow "retrograde" degeneration occurs in the root proximal to its connection with

the ganglion. That such an explanation can account for the fact that the lower cervical roots are more liable to be affected than the dorsal and lumbar is probable, owing to their shorter course, and, therefore, greater traction.

VI.

Operation can have no direct effect in causing posterior degeneration; twelve cases were operated on, and in eight degeneration was present, and in four there was no degeneration.

Age.—The ages of the patients varied from 1½ years to 64 years. Eight were under the age of 10; in six of these eight cases the tumour was situated in the cerebellum, in the seventh in the pons, and in the eighth in the corpora quadrigemina and optic thalamus. Of these eight children, in five posterior degeneration was present, while in the remaining three no degeneration could be found. It is, therefore, certain that age has no influence on the occurrence of this degeneration.

The knee-jerk.—The knee-jerks were absent in seven cases (9, 10, 14, 19, 24, 25, 27), and were diminished or absent on one side in five other cases (11, 13, 15, 22, 23).

Now, all these cases, except No. 14, showed some degeneration, and it was marked, not only in the cervical region, but also in the lumbar, in all the members of the first group, except Nos. 24 and 25.

It will be noted that of the cases with absent knee-jerks, Nos. 9, 10, 14 are tumours of the temporo-sphenoidal lobe, and that Nos. 25 and 27 are tumours of the choroid plexus and corpora quadrigemina respectively, and that Nos. 19 and 24 were cerebellar tumours.

That the presence or the absence of the knee-jerks is connected with the presence or absence of posterior degeneration in the lumbar region of the cord is, certainly proved in many of the cases here recorded, but such an explanation will not suffice, however, in every case.

The presence of the knee-jerk does not negative the possibility of posterior degeneration, for other conditions

may tend to bring about the preservation of the jerk, notwithstanding the presence of posterior degeneration in the lumbar region, *e.g.*, the occurrence of degeneration in the pyramid.

The absence of the knee-jerk in cases of cerebral tumour, when the observation is made before the supervention of coma or the last stages of the disease would seem to indicate degeneration of the posterior columns.—Case No. 14 might be cited as a case to the contrary, but it was only towards the end that the knee-jerks were abolished, and accurate observations were not made until the patient was already in a drowsy condition. Case No. 15, in the second group of cases in which the knee-jerks were diminished, is more difficult of explanation, for although degeneration was present, it was only present in the cervical region.

Special attention has been drawn to the absence of the knee-jerk in tumours of the temporo-sphenoidal lobe by Coxwell, Stephen Mackenzie, and Hughlings Jackson. In 160 cases of intra-cranial tumour where the position of the tumour was ascertained *post mortem*, occurring at the National Hospital from 1883-1897, the knee-jerks were absent in 10 per cent. of the cases, and in twelve of these sixteen cases the tumour was situated in the cerebral hemispheres, and in three in the cerebellum.

In order to determine whether the condition of posterior degeneration was evidenced clinically by a loss of the supinator and triceps-jerks, the deep reflexes were examined—(*a*) in 100 healthy persons in whom they were readily elicited without any exception; (*b*) in 120 consecutive cases of intra-cranial tumour under the care of one of us (J. S. C.) during the last three years at the National Hospital in which these phenomena had been carefully recorded, the arm-jerks were absent in twenty-five cases = 20 per cent., whereas the knee-jerks were absent in twelve cases = 10 per cent. Both arm and knee-jerks were absent on one side in two cases, and the arm-jerks were absent on one side in one case. In four cases the arm and knee-jerks were variable, sometimes present, sometimes absent, and in these cases the cerebellum was the seat of disease. From this it would appear that

consequent upon the greater frequency of the incidence of the posterior degeneration upon the cervical region, the absence of the arm-jerks in cases of intra-cranial tumours is twice as common as is the absence of the knee-jerk, and that variable deep reflexes, now present, now absent, are met with in connection with subtentorial growths.

We do not advance posterior degeneration as the cause of the loss of deep reflexes in all cases of intra-cranial tumour where such loss occurs. We have noticed sudden and persistent loss of these signs in several cases of vascular cerebral disease and in cases of cerebral tumours, and for these cases there must be some other explanation.

In connection with the symptomatology arising from the posterior degeneration, we have seen several cases of intra-cranial tumour in which marked hypotonia, severe lightning pains, and ataxy of tabetic type were present.

Optic neuritis.—This, in its early, marked, or late form of atrophy, was present in all cases except one (No. 27), and in this case there was marked degeneration of the posterior columns of the spinal cord.

Neuritis was present in nine cases in which there was no posterior degeneration, and in nineteen in which posterior degeneration occurred. And although it is possible that the two conditions may have a common cause, it cannot be assumed that when neuritis is present posterior degeneration is likewise present, nor when the former is absent the latter is likewise absent.

VII.

Conclusions.

(1) That degeneration of the posterior columns of the spinal cord frequently occurs in cases of intra-cranial tumour, *i.e.*, about 65 per cent.

(2) That such degeneration is more liable to affect the spinal cord in the cervical region than in the dorsal or lumbar regions, and is more marked in the postero-external than in the postero-internal columns.

(3) That such degeneration is of root origin, and arises from the point where the root enters the spinal cord; the posterior roots are always less affected than the posterior columns, and may show no degeneration.

(4) That such degeneration is due to traction on the posterior roots by the distension of the arachnoid, owing to the increased intra-cranial pressure, and especially such as tends to rapid dilatation of the ventricles and the sub-arachnoid space in the spinal cord.

(5) That such degeneration is independent both of the situation and of the nature of the tumour, except in so far as their liability to give rise to the preceding condition is concerned.

(6) That optic neuritis bears no relation to the occurrence of posterior degeneration.

(7) That absence of the knee-jerks (except in the semi-comatose state) indicates posterior degeneration, but that their presence does not negative such a condition, and absence of the arm-jerks has the same significance.

(8) That degeneration may occur in the direct cerebellar tract; such degeneration being directly due to pressure on the cervical region of the cord.

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GENERAL EXPLANATION OF FIGURES AND DIAGRAMS.

THE reproductions of the tumours in the brain are from photographs taken after the brain had been hardened in formalin solution.

The diagrams of the cord are from sections stained by the Marchi method, and have been drawn under an Edinger's projection apparatus.

The areas of degeneration have been indicated whilst the slide was in position, whenever this has been possible; but owing to the low magnifying power of the apparatus, it is often impossible to indicate the slighter amounts of degeneration, and these have been filled in subsequently under the microscope.

It is impossible to represent every degenerate fibre by a dot, which is relatively too large, but an endeavour has been made to keep the relative amount of degeneration in the various sections as accurate as possible.

CASE 1.

Tumour of the left frontal lobe. (Glio-sarcoma.)

Sections of the medulla and spinal cord show:—(1) Degeneration in the postero-external column in the cervical region ascending to the posterior column nuclei; (2) Degeneration in the direct cerebellar tracts above the level of the 2nd cervical; (3) Degeneration of the pyramids, most marked below the level of the 2nd cervical but present also above; (4) Degeneration in the intra-medullary portion of the 12th nerve.

PLATE I.

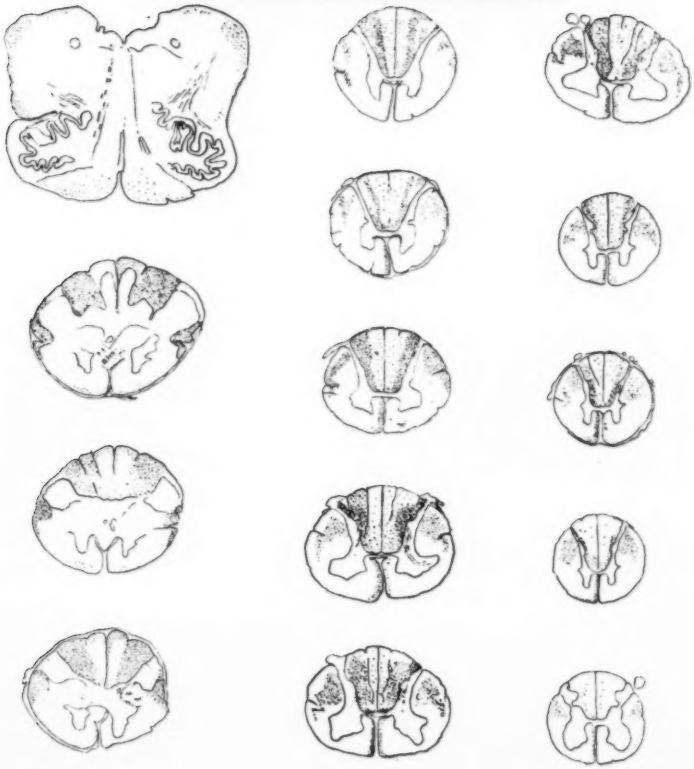
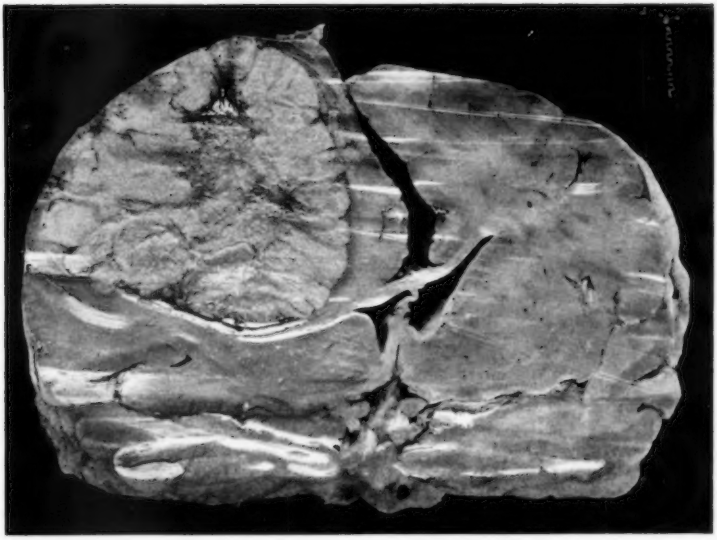


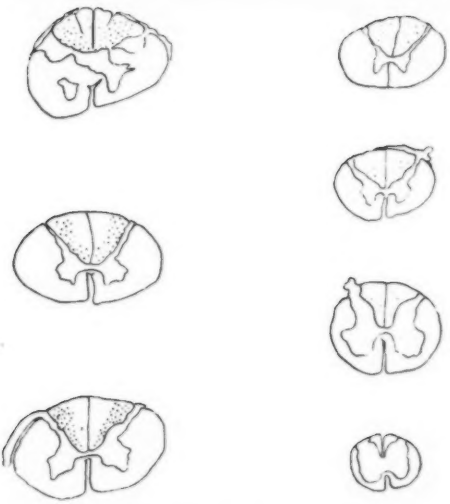
PLATE II.

CASE 2.

Tumour of the left frontal lobe. (Mixed-celled sarcoma.)
The cord shows no degeneration.

PLATE III.





CASE 4.

Tumour of the right frontal lobe. (Psammoma.)

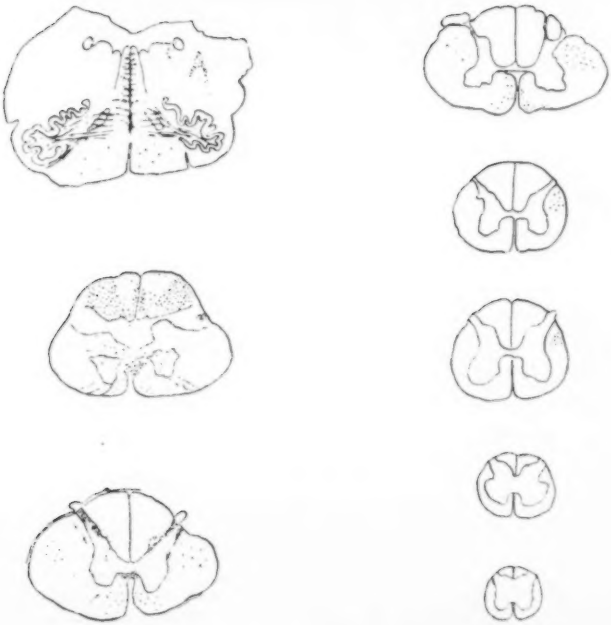
The cord shows slight degeneration in the posterior columns in the cervical region, and a few degenerate fibres are also present in the dorsal and lumbar region.

CASE 5.

Tumour of the right and left frontal and parietal regions. The tumour was removed from the right side. (Alveolar sarcoma or endothelioma.)

The cord shows degeneration in the posterior columns above the level of the 1st cervical but none below this point, although there is some degeneration in the crossed pyramidal tracts.

PLATE IV.



CASE 6

Tumour of the right frontal lobe. (Glioma.)

The degeneration in the cord is limited to the 5th left lumbar root, and this can be traced up through the dorsal to the cervical region.

There is also degeneration in the posterior root from its exit from the posterior root ganglion to its entrance into the cord. The degeneration is more marked by the Weigert-Pal method than by the Marchi method, and is evidently of long standing. It is, probably, therefore a fortuitous condition, and unconnected with the presence of the cerebral tumour.

PLATE V.

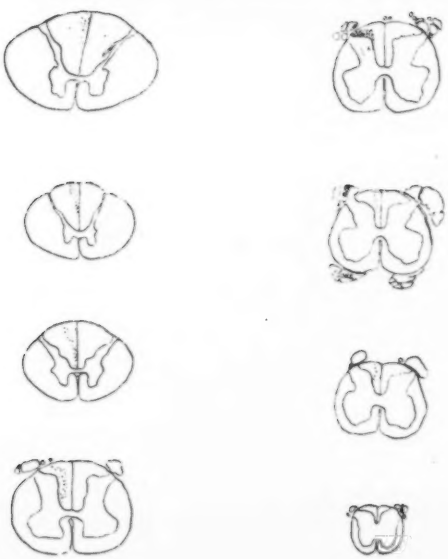
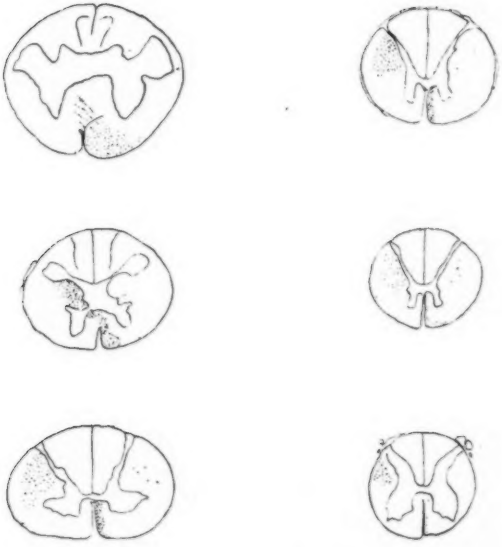


PLATE VI.



CASE 7.

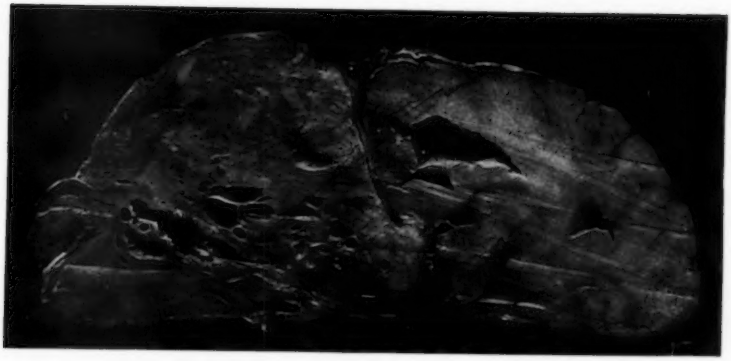
Tumour of the right motor region (removed by operation).
The cord shows no lesion of the posterior columns, but marked degeneration in the left crossed and right direct pyramidal tracts.

CASE 8.

Tumour of the right hemisphere partly removed by operation. (Mixed-celled sarcoma.) The multiple cystic condition present in the neighbourhood of the tumour is due to *post-mortem* emphysema which has occurred in this region.

The cord shows slight degeneration of the posterior columns, and marked degeneration of the left crossed and right direct pyramidal tracts and some degeneration of the opposite tracts.

PLATE VII.



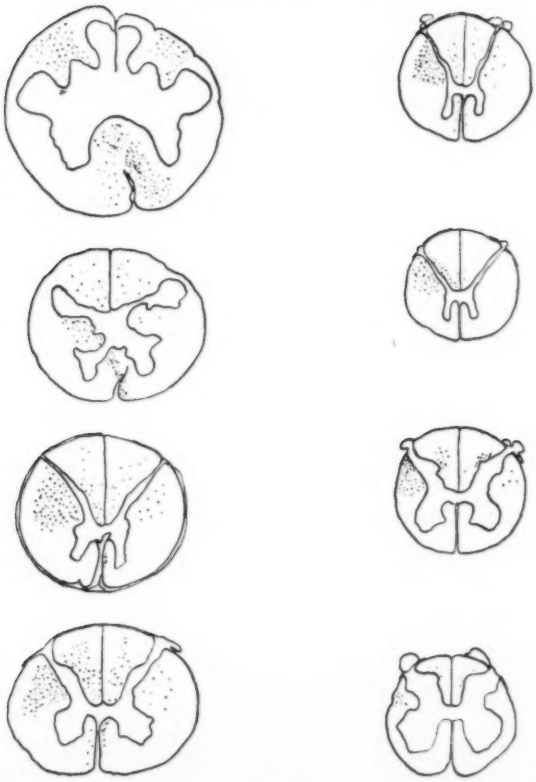
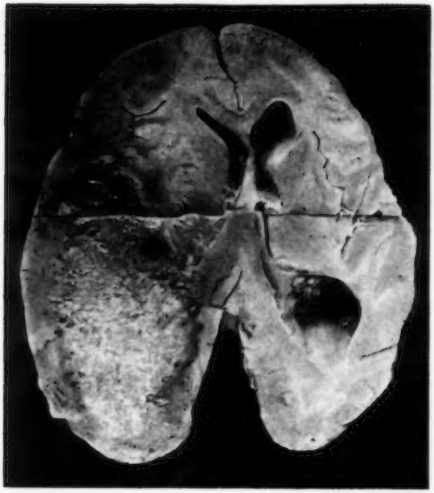
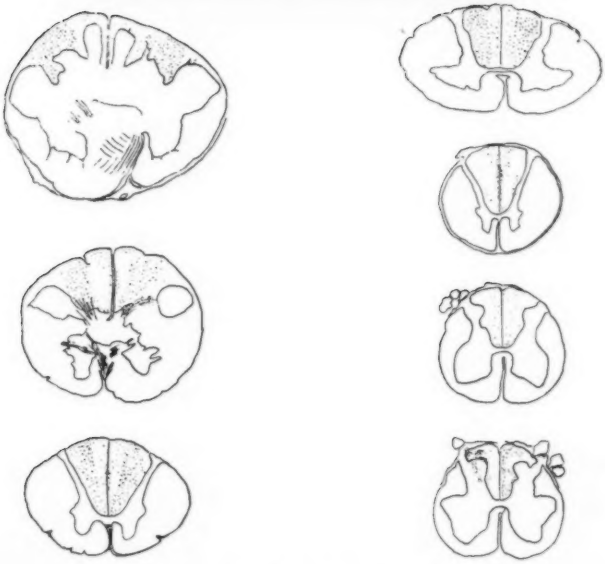


PLATE VIII.

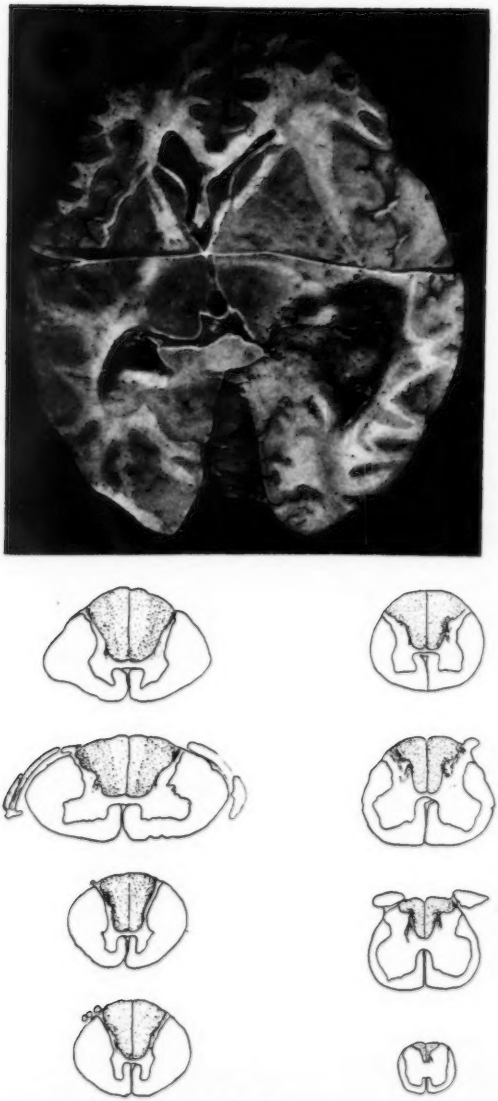




CASE 9.

Tumour of the left temporo-sphenoidal lobe. (Mixed-celled sarcoma.)
The cord shows marked degeneration of the posterior columns from the sacral to the cervical region, being most marked in the postero-external columns of the lower cervical region.

PLATE IX.



CASE 10.

Tumour of the right temporo-sphenoidal region. (Glioma.)
The cord shows marked degeneration from the sacral to the cervical region. In the cervical region the degeneration is most marked in the postero-external columns, while in the lumbar region it is evenly distributed throughout both.

CASE 11.

Tumour of the right temporo-sphenoidal region. (Spindle and round-celled sarcoma.)

The cord shows marked degeneration from the sacral to the cervical region. In the cervical region the degeneration is most marked in the postero-external columns, while in the sacral region there is degeneration around the posterior median fissure arising probably from a lower sacral root, although occupying the region of the septo-marginal tract.

PLATE X.

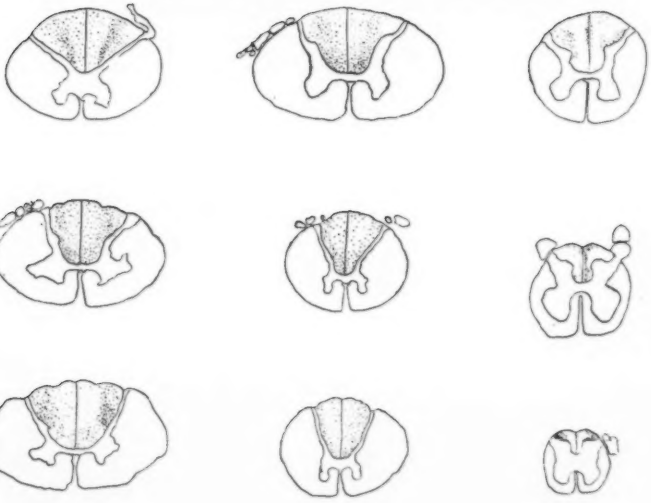
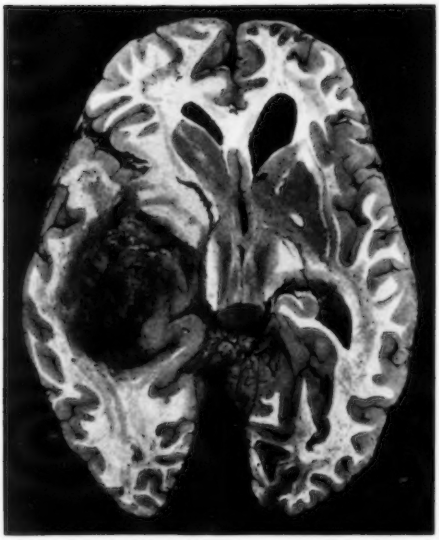
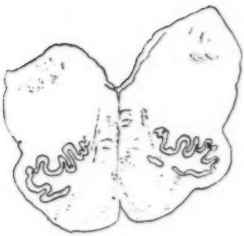
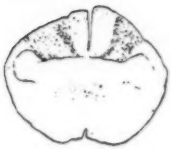


PLATE XI.







CASE 12.

Tumour of the right temporo-sphenoidal lobe. (Irregular-celled sarcoma.)
Photographed from the ventral surface.
The cord shows marked degeneration in the postero-external columns.

CASE 13.

Tumour of the left temporo-sphenoidal lobe. (Glioma.)

Only the upper portion of the tumour is shown.

There is degeneration in the posterior columns corresponding to the 4th and 5th right cervical roots, and this degeneration can be traced up to the funiculus cuneatus. There are a few degenerate fibres in the dorsal region of the cord and also in the crossed pyramidal tract of the right side.

PLATE XII.

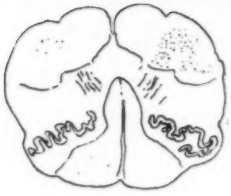


PLATE XIII.

CASE 14.

Tumour of the left cerebral hemisphere, involving the left temporo-sphenoidal lobe in its greatest extent. (Spindle-celled sarcoma.)
The cord shows no degeneration.

CASE 15.

Tumour of the left temporo-sphenoidal lobe. (Mixed-celled sarcoma.)
The cord shows degeneration of the right 6th and left 7th cervical roots, and these can be traced up to the funiculus cuneatus.

PLATE XIV.

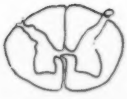
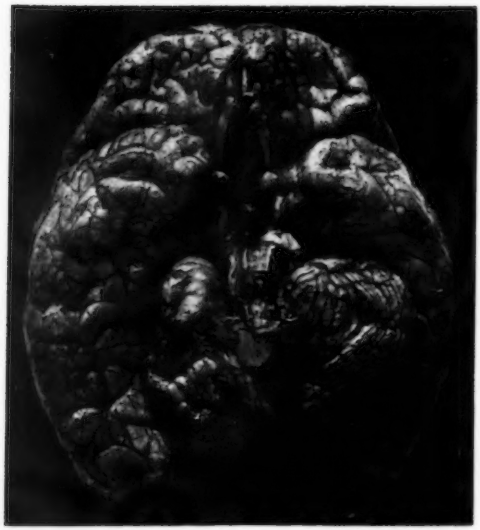


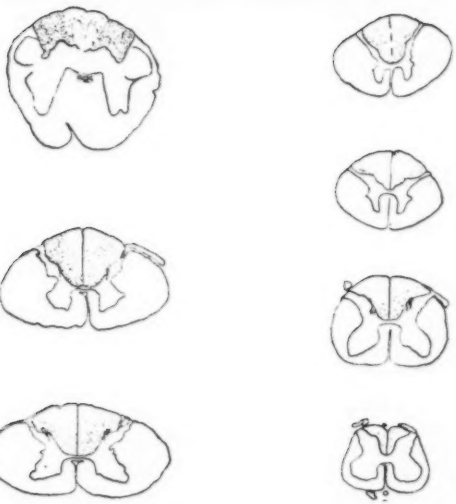
PLATE XV.

CASE 16.

Tumour of the left lobe of the cerebellum growing from the corpus dentatum and extending into the pons. (Infiltrating glioma.)
The cord appears perfectly normal.

PLATE XVI.



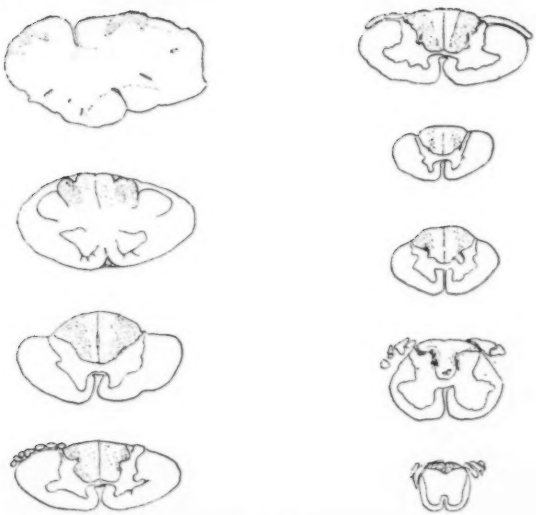


CASE 17.

Tumour of the right lobe of the cerebellum. (Fibro-sarcoma.)

In the cord there is marked degeneration of the postero-external columns in the cervical region, less degeneration in the dorsal and only a few scattered fibres in the lumbar region.

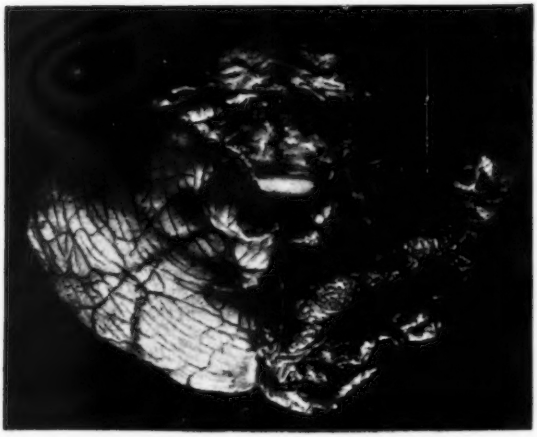
PLATE XVII.



CASE 19.

Tumour of the left lobe of the cerebellum. (Glioma.)
The cord shows degeneration in the posterior columns from the cervical to the sacral region, being however most marked in the lower cervical region.

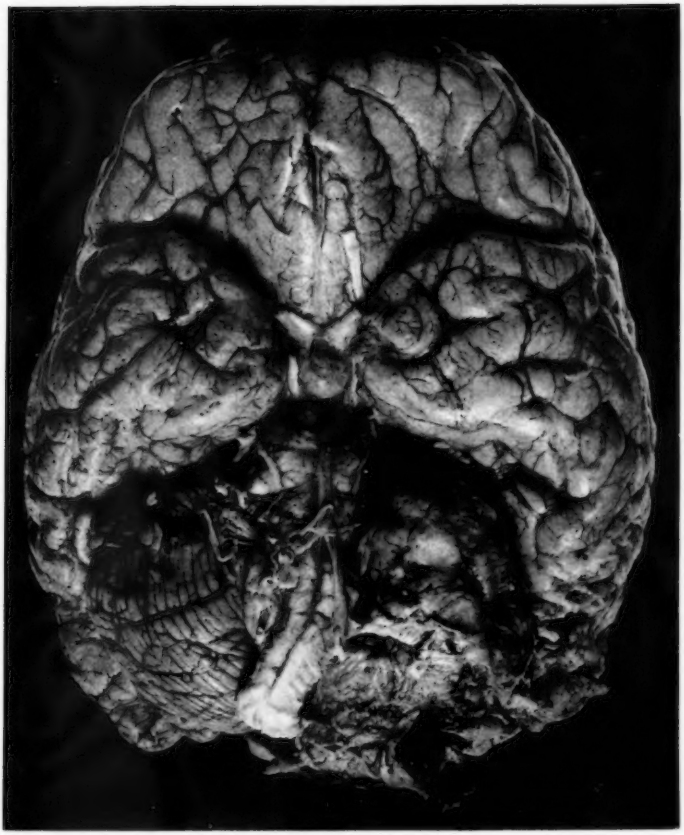
PLATE XVIII.



CASE 20

Tumour of the left lobe of the cerebellum. (Round-celled sarcoma.)
The upper portion of the cord shows degeneration of the posterior columns and of the direct cerebellar tract on both sides.

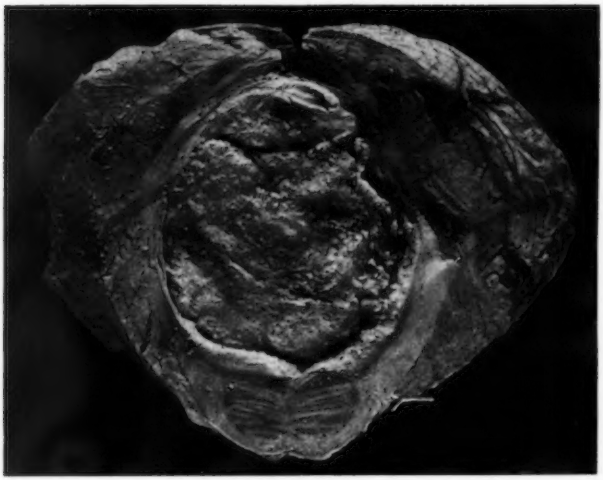
PLATE XIX.

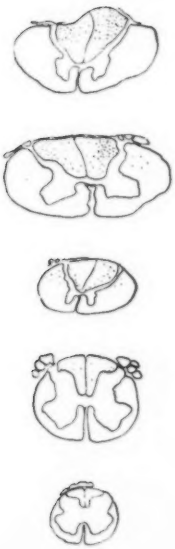


CASE 21.

Tumour of the left lobe of the cerebellum. (Fibro-sarcoma.)
The cord shows no degeneration.

PLATE XX.

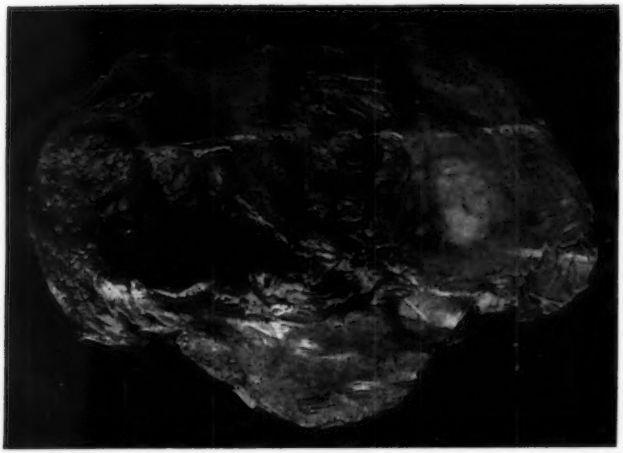


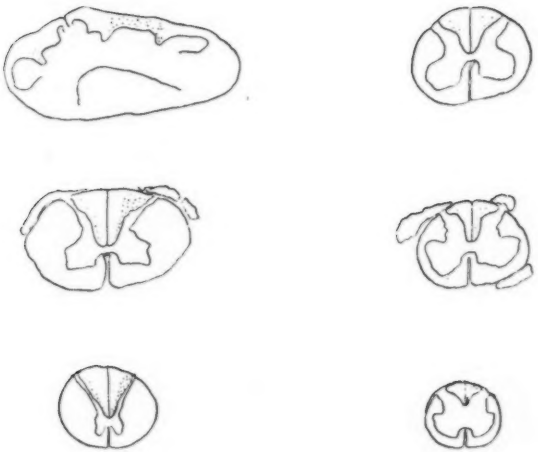


CASE 22.

Tumour of the middle lobe of the cerebellum. (Alveolar-sarcoma.)
The cord shows some degeneration of the posterior columns in the cervical region, less in the dorsal, and very little in the lumbar.

PLATE XXI.



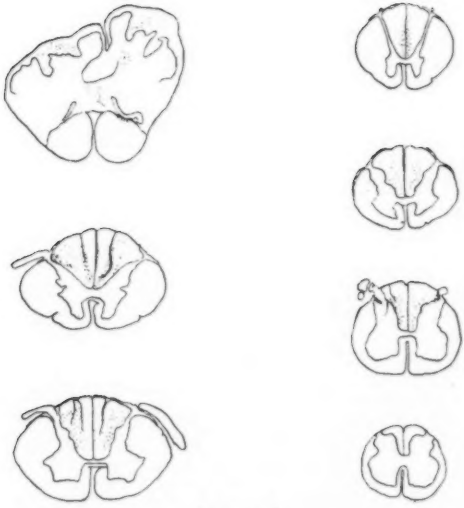


CASE 23.

Tumour of the right lobe of the cerebellum which has undergone cystic degeneration. (Glio-sarcoma.)

The cord shows slight degeneration of the posterior columns most marked in the cervical region.

PLATE XXII.

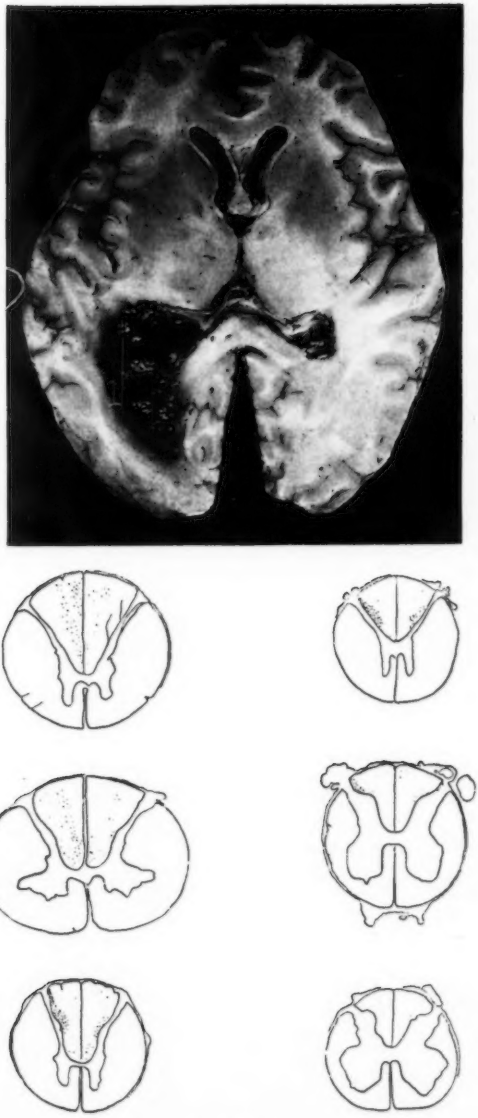


CASE 24.

Tumour of the left lobe of the cerebellum. (Glioma.)

The cord shows slight degeneration of the posterior columns, most marked in the postero-external columns in the cervical region.

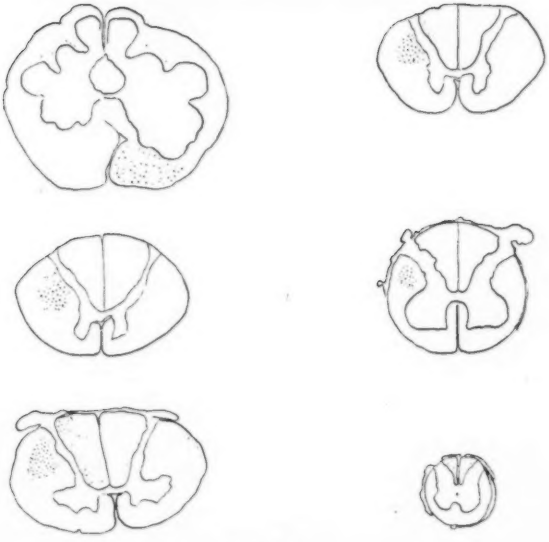
PLATE XXIII.



CASE 25.

Tumour of the choroid plexus and left lateral ventricle. (Glio-sarcoma). The cord shows degeneration in the posterior columns in the lumbar, dorsal and cervical regions.

PLATE XXIV.

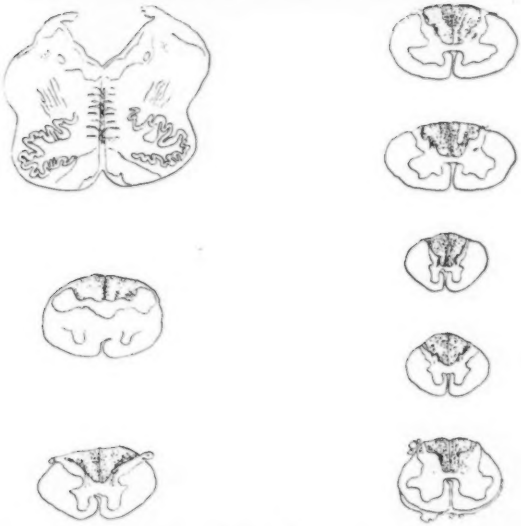


CASE 26.

Tumour of the right optic thalamus. (Tuberculous.)

The cord shows marked descending degeneration in the crossed pyramidal tract on the left side, and slight degeneration in the posterior columns in the cervical and dorsal regions.

PLATE XXV.

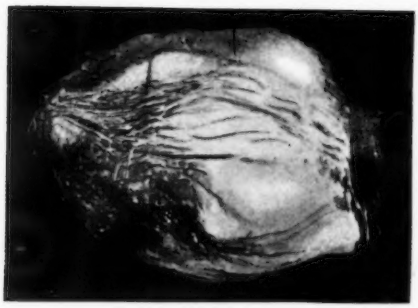


CASE 27.

Tumour of the right optic thalamus and corpora quadrigemina. (Round-celled sarcoma.)

The cord shows marked degeneration of the posterior columns from the lumbar to the cervical region.

PLATE XXVI.



CASE 28.

Glioma of the pons. The photograph is taken from a transverse section through the pons stained by the Weigert-Pal method. The transverse fibres of the pons can be seen separated by the infiltrating growth.

The cord shows no degeneration.

PLATE XXVII.

CASE 29.

Cyst of the choroid plexus growing into the 3rd ventricle.
The cord shows no degeneration.

Case.	Age.	Situation of tumour.	Size in centimetres.	Nature of growth.	Operation.	Duration of symptoms.	Optic neuritis.	Knee-jerks.	Elbow and wrist-jerks.	Degeneration of posterior columns.	Degeneration of posterior roots.	Other degeneration.	Evidence of increased intracranial pressure.	Condition of ventricles.
Frontal.														
1	30	L. frontal	5 × 5	Glio-sarcoma	O	5 mths.	Intense optic neuritis with hæmorrhage	Present and equal	Present	Marked	Marked	12th nerve; direct cerebellar pyramids	General flattening	Not dilated.
2	44	R. frontal	6 × 5·5	Mixed-celled sarcoma	O	6 wks. or 2 wks.	Intense optic neuritis	Brisk and equal	Present and equal	None	Not examined	None	Flattening R. > L.	Not dilated.
3	23	L. frontal (Broca)	$3\frac{3}{4}$ × 3	Gumma	O	$2\frac{1}{2}$ yrs.	Early optic neuritis	Present L. > R.	Brisk and equal	None	None	None	Slight flattening	Not dilated.
4	17	R. frontal	7 × 5·5	Psammoma	O	11 mths.	Intense optic neuritis	Present and equal	Present and equal	Slight	Not examined	None	Some flattening	Not dilated.
5	31	R. and L. frontal and parietal	13 × 8 × 4	Alveolar sarcoma	X	Epilepsy 10 yrs.; Hemipl. 10 mths.	Optic neuritis	Present	Present	Slight	Not examined	Both pyramids	No flattening	Some dilatation.
6	36	R. frontal	5 × 5	Glioma	X	18 mths.	Intense optic neuritis	Active R. > L.	Active and equal	5th lumbar root	5th L. lumbar root	None	Some flattening	Not dilated.
Parietal.														
7	30	R. motor	$8\frac{1}{2}$?	Not examined	X	12 mths.	Moderate optic neuritis	Active L. > R.	L. > R.	None	Not examined	R. pyramid	No flattening	Not dilated.
8	32	R. motor	9 × 7	Mixed-celled sarcoma	X	11 mths.	Optic neuritis	Present L. > R.	—	Slight	Not examined	Both pyramids	Flattening	Not dilated.
Temporo-sphenoidal.														
9	15	L. temporo-sphenoidal	10 × 5	Mixed-celled sarcoma	O	10 mths.	Intense optic neuritis	Present at first; absent later	Present at first; absent later	Moderate	Marked	None	Flattening	Dilated.
10	12	R. temporo-sphenoidal	5 × 4	Glioma	O	6 mths.	Intense optic neuritis	Absent	Absent	Marked	Marked	Lissauer's tract	Marked flattening	Some dilatation.
11	21	R. temporo-sphenoidal	7 × 7	Spindle and round-celled sarcoma	X	18 mths.	Post-neuritic atrophy	Not easily obtained	Absent	Marked	Very slight	Lissauer's tract; ascending root of 5th fillet	Flattening	Not dilated.
12	34	R. temporo-sphenoidal	6 × 6	Irregular-celled sarcoma	X	4 mths.	Intense optic neuritis	Present L. > R.	Absent	Marked	Not examined	Fillet; cerebellar peduncle pyramid	Marked flattening	Somewhat dilated.
13	64	L. temporo-sphenoidal	5 × 3 and 2 × 2	Glioma	O	6 wks.	Slight optic neuritis	R. just obtained; L. absent	Absent	Moderate	None	Pyramid	Some flattening	No dilatation.
14	50	L. cerebral hemisphere and crus	5·5 × 8	Spindle-celled sarcoma	O	2 mths.	Optic neuritis	R. not obtained; L. present	R. absent; L. present	None	Not examined	None	Flattening	No dilatation.
15	50	L. temporo-sphenoidal	6 × 5	Mixed-celled sarcoma	O	10 mths.	Optic neuritis	R. present; L. difficult to obtain	Present and equal	Slight	Not examined	None	Slight flattening	Slight dilatation.
Cerebellar.														
16	7	Middle lobe of cerebellum	Infiltrating	Glioma	X	$3\frac{1}{4}$ yrs. ?	Post-neuritic atrophy	Brisk	Brisk and equal	None	Not examined	None	No flattening	Marked dilatation.
17	47	R. lateral lobe of cerebellum	3 × 5	Fibro-sarcoma	X	3 yrs. ? 1 yr.	Intense optic neuritis	Present and equal	—	Marked	Not examined	None	No flattening	—
18	5	L. lateral lobe of cerebellum	?	Tuberculous	X	8 mths.	Intense optic neuritis	Present and equal	Present and equal	None	Not examined	None	Flattening	Very dilated.
19	5	L. lateral lobe of cerebellum	7 × 4	Glioma	O	8 mths.	Intense optic neuritis	Present at first; absent later	—	Marked	Marked	None	No cerebral flattening, but subtentorial pressure	Not dilated.
20	6	L. lateral lobe of cerebellum	7 × 3·5	Small round-celled sarcoma	X	4 mths.	Intense optic neuritis	Active at times; absent at others	Present	Marked	Not examined	Direct cerebellar tract	Marked flattening	—
21	33	L. lateral lobe of cerebellum	6 × 4·5	Fibro-sarcoma	X	3 yrs. and 3 mths.	Optic atrophy	Present and equal	—	None	Not examined	None	No flattening	Dilated.
22	7	Middle lobe	5 × 5	Alveolar sarcoma	O	4 mths.	Optic neuritis	Diminished	Diminished	Moderate	Not examined	Pyramids	Marked flattening	Marked dilatation.
23	30	R. lateral lobe	4 × 3·5	Glio sarcoma	O	19 mths.	Intense optic neuritis	R. sometimes absent	Diminished	Slight	Not examined	None	Not stated	Not stated.
24	6	L. lateral lobe	4·5 × 3	Glioma	X	7 mths.	Optic neuritis	Absent	Absent	Slight	None	None	Marked flattening	Dilated.
Basal ganglia, ventricles and pons.														
25	24	Choroid plexus and L. ventricle	5 × 3	Glio-sarcoma	O	8 mths.	Intense optic neuritis	Absent	Absent	Moderate	Not examined	None	No flattening	Dilated by growth.
26	27	R. optic thalamus	4·5 × 4·5	Tuberculous	O	2 yrs.	Optic neuritis	Present L. > R.	Present L. > R.	Slight	Not examined	Pyramid	Marked flattening	Marked dilatation.
27	$1\frac{7}{12}$	R. optic thal. and corp. quad.	2 × 4	Round-celled sarcoma	O	9 wks.	No neuritis	Absent	—	Marked	None	Fillet; posterior long. bundle; sup. ped. of cerebellum	Marked flattening	Marked dilatation.
28	7	Glioma of pons	Infiltrating	Glioma	O	11 dys.	Optic neuritis	Present	—	None	Not examined	None	Marked flattening	Marked dilatation.
29	42	Cyst in 3rd ventricle	1·5 × 1·5	Cyst of choroid plexus	O	14 dys.	Discs hyperæmic	Present	Present	None	Not examined	None	Marked flattening	Marked dilatation.

List of Published Cases.

Sex.	Age.	Situation of tumour.	Nature.	Duration.	Optic neuritis.	Knee-jerks.	Degeneration of posterior columns.	Degeneration of posterior roots.	Other degeneration.	Author.
F. 1894	33	L. cerebral hemisphere	Glioma	5 mths.	Present	Absent	Marked; cervical +	No degeneration	Cells of anterior horn	Mayer
M. 1894	46	Pressure on both frontal lobes	Endothelioma	12 mths.	Present	Absent	Marked; cervical +	None	None	Mayer
F. 1895	4	R. cerebral hemisphere	Spindlé-celled sarcoma	7 mths.	Present	Absent	Marked; cervical +	Present	None	Dinkler
1890	—	L. lobe of cerebellum	—	—	—	—	Marked	—	Ascending root of 5th L. cerebellar tract	Wollenberg
F. 1894	62	Softening of L. cerebellar lobe	Softening	2 yrs.	—	—	Marked	—	L. pyramid	Campbell
M. 1894	73	L. lobe of cerebellum	Softening	?	—	Diminished	Marked; cervical +	—	L. sup. med. and inf. peduncle	Campbell
M. 1897	53	L. cerebral hemisphere	Spindle-celled sarcoma	5 mths.	None	Obtained	Marked; cervical +	Present	—	Ursin
M. 1897	60	L. occipital and R. frontal	—	5 mths.	Present	Active	Marked; cervical +	Present	—	Ursin
M. 1897	67	L. hemisphere	Cystic	4 mths.	Not examined	Active	Marked	None	Hypo-glossal on R. side	Ursin
M. 1896	47	L. hemisphere	Not stated	Over 2 mths., not def. stated	Present	Active R. > L.	Marked	Present	Pyramidal tracts Pyramid	Pick
M. 1896	50	R. hemisphere	Not stated	16 mths.	Active	K.-J. active	Marked; cervical +	—	—	Pick
C. 1896	5	Osteo-sarcoma of skull	Sarcoma	Not stated	Not stated	Not stated	Marked; cervical +	None	—	Pick
F. 1896	68	Both cerebral hemispheres	Glioma	Not stated	None	Active at first Diminished R. > L.	None	—	—	Pick
F. 1896	38	Diffuse cerebral sclerosis	—	11 mths.	Present	R. absent L. difficult	Marked; cervical +	None	—	Pick
F. 1896	16	3rd ventricle	—	18 mths.	Atrophy	Active	Marked; cervical +	None	Gowers' tract	Besold
F. 1896	11	Optic thalamus	—	3 yrs.	Atrophy	Active	Marked; cervical +	None	Gowers' tract	Besold
M. 1897	20	R. cerebral hemisphere	—	19 mths.	Present	Active	Marked	None	Left pyramid	Hoche
F. 1897	49	L. lobe of cerebellum	Glio-sarcoma	1 yr.	Present	Diminished	Marked; cervical +	None	—	Hoche
F. 1897	33	L. cerebral hemisphere	Fibro-sarcoma	3 mths.	None	Exaggerated	None	—	—	Hoche
M. 1899	19	Injury to R. cerebral hemisphere	—	20 days	—	-	Marked; cervical +	—	—	Bikeles

Appendix.

Table of Cases.

Frontal 1, 2, 3, 4, 5, 6
Parietal 7, 8
Temporo-sphenoidal 9, 10, 11, 12, 13, 14, 15
Cerebellar 16, 17, 18, 19, 20, 21, 22, 23, 24
Basal Ganglia and Pons 25, 26, 27, 28, 29

Case 1.—J. H., a lady's maid, aged 32 years, was admitted into the National Hospital under the care of Sir William Gowers, on July 15, 1898, complaining of headache, vomiting and dimness of sight. Her family history was unimportant. With the exception of some attacks of neuralgia in the forehead and two slight attacks of influenza, her health had always been excellent.

In March 1898, after an attack of sea-sickness, she first noticed severe headache "behind the eyes" and pain running down the back of the neck. The headache continued and increased in severity and she vomited daily for three months; the emesis bearing no relation to meals.

Two months later, she constantly saw double, and noticed that her sight was getting dim. No other symptom was complained of.

When admitted, she was healthy-looking and of very good intelligence. She stated that she was losing her memory, and that but for the headache and double vision she felt perfectly well.

Taste, smell and hearing were normal.

Vision $\frac{6}{5}$, J, with either eye. Visual fields and colour vision were good. Intense double optic neuritis with many hæmorrhages and much exudation.

Pupils normal. Weakness of right external rectus with corresponding diplopia.

Motion, gait, objective sensation, normal. Knee, wrist and elbow-jerks present and equal; plantars sluggish.

On June 19, on waking in the morning, she began to talk noisily and incoherently and had varied delusions. Face flushed, pupils dilated R. > L. No mental aberration had been present till this time. The delusions were present for three days.

On June 28 the following note was made: No delusions. Mental state normal. Pain much less. Visual fields markedly contracted. Weakness of both external recti. No other symptom had appeared, knee-jerks present.

July 7. She was much better. Mental state good. Headache slight. No fresh symptoms. She was sitting up and about the ward.

July 18. Much worse, no further symptoms. Mental state petulant and childish.

August 20. Much worse, and mentally heavy and dull. Knee-jerks present and equal. No further symptoms.

August 23. Difficulty of swallowing. She gradually became comatose, and died of respiratory failure.

Duration of symptoms five months.

Description of tumour (plate i.).—There was general flattening of the convolutions. The anterior portion of the falx was infiltrated by new growth and adherent to the superior frontal convolution. A large tumour was present in the left frontal lobe and was of a dark reddish colour with small dark areas within it. The growth was irregularly circular, and measured about 5 cm. in diameter, it had a sinuous outline and sharply defined limits except posteriorly where it merged into the normal brain tissue. The tumour had made its way across the middle line and was pressing on the inner border of the opposite hemisphere.

Microscopical examination (plate i.).—At the level of the corpora quadrigemina there was a small area of softening just at the outer angle of the fillet.

In a section taken through the middle of the pons, a bundle of degenerate fibres can be seen on the outer side of the fillet passing in a dorsal direction. There were some scattered degenerate fibres in the posterior longitudinal bundles, and in the pyramids.

At the level of the olive there was marked degeneration in both restiform bodies, and to a slight degree in both pyramids. The twelfth nerve was also degenerate on one side. The fibres of the fillet were practically free from any degeneration.

At the level of the posterior column nuclei there was marked degeneration in both the funiculus gracilis and cuneatus, and this was more marked in the latter than in the former. Marked degeneration was also present in the direct cerebellar tracts of both sides. The pyramids showed very little degeneration.

In the upper cervical region of the cord there was very marked degeneration in the postero-external and postero-internal columns of the cord and also in the direct cerebellar tract, but the pyramids showed very little degeneration. Below the level of the second cervical root there is very little degeneration in the direct cerebellar tract, but in its place there is a band of

degenerate fibres lying between this tract and the pyramidal tract and this band extended forwards into the antero-lateral tract.

In the lower cervical region the degeneration in the posterior columns was still more marked, and that in the pyramidal tracts seemed rather to increase than diminish when traced downwards to the mid-dorsal region, but below this point the degeneration in the pyramidal tracts became progressively less marked.

In the lumbar region there was slight degeneration in the posterior columns and also in the crossed pyramidal tracts.

The degeneration extends into the posterior roots in the cervical region, but it is by no means so marked as that in the posterior columns.

By the Weigert-Pal method the cord appeared normal, and Clarke's column and Lissauer's tract were also normal.

Nature of growth.—The growth was composed of cells of varying shapes and sizes, some being triangular with a fine prolongation, and they were rather closely packed together—the prolongations of the cells forming a matrix in which the cells lay. The growth was not very vascular and in the portion cut seemed fairly uniform in structure (glio-sarcoma).

Case 2.—K. N., aged 44, was admitted into the National Hospital under the care of Dr. Beevor, on October 3, 1899.

The patient had complained of headache for six weeks, worse at night and paroxysmal in character, and he had vomited on one occasion. Three weeks before admission he went to a convalescent home and while there became dull and stupid, his eyesight began to fail and he staggered. He had fallen on two occasions hitting the left side of his head.

On admission he was very stupid, fatuous and very emotional. He could answer questions but not always correctly, and his articulation was very indistinct. He could name objects, but appeared unable to read printed or written words. He could write to dictation but could not copy nor could he read what he had written. Both optic discs showed intense optic neuritis. There was no loss of motor power or ataxy in the limbs or trunk, but the gait was unsteady and he tended to fall backwards and to the left. The knee-jerks were brisk and equal, and there was no ankle clonus. The arm-jerks were normal and equal, the plantar reflexes were lively and equal. Three days after admission he rapidly became comatose; the limbs became flaccid; the knee and arm-jerks could not be obtained and he died on October 6.

Description of tumour (plate ii.).—There was flattening of the convolutions, and situated in the left frontal was a tumour occupying the second and adjacent parts of the first and third frontal convolutions. The dura mater was adherent over its surface. On section an oval tumour measuring 6 cm. in the antero-postero diameter, and 5·5 cm. in the transverse direction, was seen reaching almost to the middle line, but not extending far enough back to affect the ascending frontal convolution.

Microscopical examination.—The cord appeared perfectly normal by the Marchi, Weigert-Pal and by the blue-black methods.

Nature of growth.—The growth was composed of large cells of irregular shape and size, among which were situated areas of a substance which took the stain badly, and in these regions the cells were broken up into a kind of *débris*. The growth was not vascular, and the coats of the vessels surrounding the growth were thickened. The appearance of the growth did not suggest a gumma but a mixed cells sarcoma undergoing degeneration. (Mixed celled sarcoma.)

Case 3.—B. K. was admitted to the National Hospital under the care of Dr. Bastian, on November 14, 1898. Two and a-half years previous to admission she suddenly fell down in the street and lost consciousness for a few minutes, and during the following fortnight she had two fits of a similar nature. Since that time she had had no more fits but had suffered from attacks of giddiness, for which she attended the out-patient department for eighteen months before admission. During the month of October, 1898, she was noticed to be mentally dull, and in the early part of November it was noticed that she used wrong words and complained of slight headache. On the morning of November 13 she was very drowsy, and on the following day was admitted to the hospital.

On admission the patient was found to be a well-nourished, healthy-looking girl. She lay in bed in a semi-comatose condition. There was complete paralysis of the right sixth nerve and nearly complete paralysis of the left sixth nerve. There was recent optic neuritis in both eyes. The left lower face did not move so well as the right. Motion and sensation in the trunk and limbs were natural. The knee-jerks were not exaggerated, the left being rather more easily obtained than the right. The elbow and wrist-jerks were brisk and equal. The plantar reflexes gave a flexor response. There was no aphasia. She rapidly became comatose and died seven days after admission. (The

case was fully reported by Dr. Collier in the *Lancet*, March 25, 1899.)

Description of tumour.—The dura mater was adherent to the anterior part of the left temporo-sphenoidal lobe, the lepto-meninges at the base of the brain showed considerable thickening and there was recent meningitis over the posterior extremities of the second and third left frontal convolutions. A growth was found involving the inferior margin of the second left frontal convolution, and the whole of the posterior part of the third left frontal convolution and also the anterior part of the first left temporo-sphenoidal convolution. The growth measured 3·7 by 3 cm.

Microscopical examination of the cord.—The cord appeared perfectly normal by the Marchi and Weigert-Pal methods. No degeneration was present in the posterior roots.

Nature of growth.—The growth was composed of small round cells staining deeply, and areas evidently undergoing degeneration in which little or no structure was visible. There were rounded areas with definite walls filled with what appeared to be young fibrous tissue, and some of these contained badly formed giant cells. They were probably thrombosed vessels in which organisation of the clot has taken place. The walls of the vessels were very much thickened. (Gumma.)

Case 4.—L. L., a servant, aged 17 years, was admitted into the National Hospital under the care of Dr. Hughlings Jackson, on January 12, 1899. When in apparent good health in July, 1898, she had a fit commencing in both hands, consciousness being lost late. A week later a similar fit occurred. In August, 1898, severe headache and vomiting set in. These symptoms improved greatly under treatment with mercury and iodide of potassium.

On admission she was intelligent and her memory was good. Vision $\frac{6}{60}$ badly, and Jaeger 4 with either eye. Intense double optic neuritis. Marked spontaneous nystagmus increased on lateral deviation.

Cranial nerves, motion, gait, sensation and sphincters were normal. Knee, wrist, and elbow jerks were present and equal. She began to develop slight signs of mental weakness, but no other signs appeared up till the time of her death, from a sudden attack of coma, on June 28, 1898.

Duration of symptoms, eleven months.

Description of tumour (plate iii.).—On the inner side of the skull, situated over the centre of the tumour was an exostosis of

pyramidal shape, which had caused a depression in the dura mater. There was some flattening of the convolutions, and the dura mater was firmly adherent over a large tumour situated in the right hemisphere extending from the middle line and involving the superior and middle frontal convolutions in their posterior part. The greater portion of the tumour lay anterior to the ascending frontal convolution, but one small nodule extended into it. A vertical section made through the tumour showed that it extended deeply into the cerebral substance. The corpus callosum was pushed downwards and the tumour bulged into the longitudinal fissure, causing a depression in the opposite hemisphere. The lateral ventricle of the right side was pressed upon, but there was no distention of either ventricle. The fibres of the internal capsule had escaped direct involvement by the tumour.

Microscopical examination (plate iii.).—By the Marchi method there was practically no degeneration in the sacral, lumbar and lower dorsal region of the cord. In the upper dorsal region there was slight degeneration, which increased in the cervical region. The degeneration was almost entirely confined to the postero-external column, and could be traced up to the nucleus cuneatus, where it ceased and sections above this level showed no degeneration.

By the Weigert-Pal method the cord appeared perfectly normal, and by the Nissl method no abnormality could be detected either in the cells of the anterior horn or of Clarke's column.

Nature of tumour.—The tumour was composed of spindle cells with large nuclei and these cells had an irregular alveolar arrangement, numerous cell nests being scattered irregularly throughout. The growth was a psammoma.

Case 5.—J. T., a compositor, aged 32 years, was admitted into the National Hospital under the care of Dr. Bastian, on December 5, 1898. Jacksonian attacks of the right face had been present for ten years. In May, 1898, an attack of left sided convulsions followed by transient left hemiplegia, occurred. Frequent left sided attacks then followed and he gradually became hemiplegic on the left side and also weak on the right side. Some slight headache had been present for a year.

On admission the condition was as follows:—Mental state intelligent. Optic neuritis of long standing. Marked left hemiplegia (leg and foot most affected). Slight right hemiplegia (leg and foot most affected). Trunk muscles very weak. No

anæsthesia. Knee, wrist and elbow jerks present. L. > R. Left foot clonus. There was a large hard swelling about the size of an egg, and continuous with the skull, most prominent at about the middle of the sagittal suture, and tender to hard pressure.

He had Jacksonian attacks, beginning sometimes in the left foot and sometimes in the muscles of the left side of the abdomen. Later on he had similar attacks beginning in the right foot.

An operation was performed, and a large amount of growth was removed, but the tumour which was situated in the great longitudinal fissure was too extensive for extirpation. He died on February 18, 1899.

Duration of symptoms: epilepsy ten years; hemiplegia ten months.

Description of tumour (plate iv.).—A large opening was present in the skull, extending across the middle line of the vertex, this portion of the calvarium having been removed at the time of operation. The longitudinal sinus was blocked by a mass of growth. On the right side of the brain there was a large softened area corresponding to the tumour removed, and on the opposite side close to the middle line was another mass of growth evidently of the same nature. The dura mater was adherent to the brain around the edges of the wound. A transverse section was made across the brain and this showed a large tumour involving and growing from the dura mater extending for 4 cm. into the left hemisphere immediately to the left of the longitudinal fissure. On the right side the growth did not extend so deeply, being only about 3 cm. from the surface (a large portion of the growth had been removed from this situation by operation). The growth was firm and hard, and was not very vascular. It was definitely limited and did not infiltrate the brain. The growth measured 13 cm. in longitudinal direction, 8 cm. in a transverse direction, and 4 cm. in its greatest depth, and involved both the frontal and parietal lobes.

Microscopical examination (plate iv.).—By the Marchi method the cord and medulla showed descending degeneration in the crossed pyramidal tracts of both sides, and also in the direct pyramidal tract, but this was more marked on the left side than the right; the degeneration could be traced down to the lumbar region. There was no posterior degeneration in the cord below the level of the second cervical roots, but at this level two degenerate roots could be seen entering the cord. In the segment above this there was marked degeneration both in Goll's column and Burdach's column, and this could be traced

up to the medulla. From the affection of both tracts it would seem probable that damage to the cervical cord was wider than indicated by the section at that level. Degeneration of the anterior roots also was present at that level. Some degenerate fibres were present in the raphe in the upper portion of the medulla.

By the Weigert-Pal method the posterior columns of the cord at the level of the second cervical were very pale, but no change could be found in the lower portion of the cord. When stained by aniline blue black, the cells of the anterior horn and of Clarke's column appeared normal. At the level of the second cervical there was an increase of fibrous tissue around the posterior fissure, but not affecting so wide an area as that shown by the Weigert-Pal method.

Nature of growth.—The tumour consists of spindle cells with large nuclei and irregular and indefinite alveoli. (Alveolar sarcoma.)

Case 6.—J. W., aged 26, was admitted into the National Hospital under the care of Dr. Ormerod, on August 15, 1899.

For the two years previous to admission, the patient had suffered from fits followed by attacks of vomiting, and for the last thirteen months of this period he had had otorrhœa. He had had syphilis two to three years ago. He attributed his illness to a blow on the ear two and a-half years ago, but he said that he had been deaf previously to this.

On admission he was a big muscular man with a defective memory and a confused mental state. There was extreme double optic neuritis. Hearing was defective in the left ear. He could only stand with difficulty, and tended to fall to the left and backwards when his eyes were closed, but he could, however, walk fairly straight. He was a left-handed man. All movements were well performed, but the right hand was stronger than the left. Sensation was normal. Reflexes: The elbow jerks were increased. The knee-jerks were increased and the right was somewhat greater than the left. There was no ankle clonus, and the plantars gave a flexor response. There was some tenderness over the left mastoid. The temperature was subnormal. The patient was trephined over the left temporo-sphenoidal lobe, but nothing was found, and he died on the following day.

Description of tumour (plate v.).—There was an opening in the skull over the left temporo-sphenoidal lobe. The convolutions were flattened. There was considerable destruction of the

left temporo-sphenoidal lobe due to the operation. The right superior frontal convolution was broad and flattened, and on palpation felt hard, and it was obvious that a large tumour occupied the right frontal region, and it could be seen bulging into the longitudinal fissure and compressing the cerebral hemisphere of the opposite side. On section, a cystic tumour containing yellowish gelatinous material was found to occupy the right frontal region. The tumour measured 5 × 5 cm., and appeared to have a fairly definite margin. Besides a large cyst there existed numerous smaller ones situated in the peripheral portion of the growth. The growth extended across the middle line and lay between the corpus striatum and the corpus callosum. The basal ganglia on the right side were pressed backwards. The grey matter of the cortex in the frontal region was very thin and presented no clear line of demarcation from the white matter.

On careful inspection of the spinal cord it was seen that the fifth left lumbar root was grey and thin as compared with the corresponding root on the other side. No other root appeared to have been affected.

Microscopical examination (plate v.).—By the Marchi method there was degeneration of the fifth left lumbar root and the degenerate fibres from this could be seen at the level of the third lumbar root. The number of degenerate fibres, as seen by this method, was comparatively small when compared with the marked degeneration as shown by the Weigert-Pal method.

The degeneration could be distinctly traced up to the mid-dorsal region, above which, however, it becomes very little marked, while at the level of the second cervical root there was practically no degeneration. There was no degeneration in the funiculus gracilis and cuneatus.

Weigert-Pal. By this method the degeneration of the fifth left lumbar root was well seen, and a band of degeneration could also be seen at the level of the third sacral. The lower segments however, showed no change. In the mid-dorsal region the column of Goll was distinctly paler on the left side than the right, but both the columns of Goll are paler than the columns of Burdach. In the cervical region the same condition was present, and the same change could be seen in the funiculus gracilis. No distinct change could be detected in Lissauer's tract.

By the Nissl method the cells of the anterior horn and of Clarke's column appeared normal. At the level of the third

lumbar root the cells were fewer on the left side than the right, but this condition was not striking.

An increase in the glia tissue about the degenerate root could also be seen by this method.

Nature of growth.—The growth was composed of a loose meshwork with irregular nuclei scattered sparsely throughout its substance. There were large areas of but slightly stained tissue, often surrounded by nuclei. These were probably portions of the tumour undergoing hyaline degeneration, as they closely resembled the hyaline tissue seen in the most degenerate portion of the tumour. (Glioma.)

Case 7.—W. S., aged 30, a dairyman, admitted into the National Hospital under the care of Dr. Beevor on July 18, 1898. He had been suffering from Jacksonian attacks in the left arm and face with progressive brachio-facial monoplegia for eleven months; headache and vomiting seven months; mental derangement two months.

On admission he was drowsy and delirious. Moderate double optic neuritis. Conjugate deviation to right. Marked brachio-facial monoplegia of left side. Left leg slightly weak. Anæsthesia of cortical type in left upper limb. Intense pain and great tenderness immediately above the right ear. Wrist and elbow-jerks L. > R.; knee-jerks brisk L. > R.; left foot clonus.

On July 24, Mr. Horsley removed a large tumour from the right motor area. The wound healed by first intention. The patient became maniacal and died suddenly on August 29.

Duration of symptoms twelve months.

Description of tumour (plate vi.).—There was an opening into the skull on the right side, and a large gap in the cortex of the same side, measuring about 8·5 cm. across; inferiorly, it involved the superior temporal and hinder part of middle temporal convolutions. A horizontal section into the hemisphere showed that the external capsule and the outer part of the lenticular nucleus had been removed, but that the internal capsule had itself escaped in its entire extent.

Microscopical examination (plate vi.).—Marchi's method showed recent well-marked degeneration in the right pyramid, above the decussation and in the corresponding direct and crossed pyramid below that point. No degeneration was present in the posterior columns. By the Weigert-Pal method the cord appears perfectly normal.

Growth not examined.

Case 8.—R. B., aged 32, was admitted to the National Hospital, under the care of Dr. Tooth, on December 10, 1897.

Two months before admission he had had a fit, commencing in the left great toe, spreading to the hip; this lasted ten minutes, the arm being unaffected. There was no loss of consciousness. On the following day the patient had had two similar attacks, in the second of which he lost consciousness. The fits have recurred every seven to ten days. After the third fit there was weakness of the left arm and leg. Headache had been present for two to three weeks, but there had been no vomiting.

On admission there was optic neuritis, weakness of the left side of the body, and slight left hemianæsthesia. The left knee-jerk was greater than the right; there was ankle-clonus on the left side. The optic neuritis and the weakness increased, and in February Mr. Horsley removed a large tumour from the right motor cortex. In April he could walk with assistance, and went home for a time. He returned again in June, with a hernia cerebri, and the wound was again opened up. He died about a month later, the hernia still pressing through the opening in the skull.

Description of tumour (plate vii.).—A large opening was present in the skull on the right side, the result of the operation. Through this opening a large fungating mass of brain substance protruded; the convolutions were much flattened. The right crus was much flattened and diffluent. The cerebellum and pons were normal. The brain was cut by transverse section, and a large tumour, measuring 9 cm. in the antero-posterior diameter, by 7 cm. in the lateral diameter, was seen to occupy the whole of the middle third of the right hemisphere. The tumour extended from the surface to the longitudinal fissure, and involved the falx cerebri and the hemisphere of the opposite side to a slight degree. The tumour was firm, hard, and white in appearance, with numerous irregular cavities situated throughout its substance. The margins of the tumour were fairly clearly defined.

Microscopical examination (plate vii.).—By the Weigert-Pal method, sections of the medulla and spinal cord showed very marked degeneration in the left crossed and the right direct pyramidal tract; the degeneration extended down to the lumbar region; no degeneration could be seen in the opposite pyramid by this method. By the Marchi method, the most marked degeneration was in the crossed pyramidal tract of the right side, *i.e.*, on the opposite side to the most marked degeneration, as shown by the Weigert-Pal method. There were a few degene-

rate fibres on the left side and also in the posterior columns; such degeneration being scattered and rather more marked in the cervical than in the dorsal or lumbar regions.

Nature of growth.—The growth was composed of cells of varying size and shape, among which strands of newly-formed connective tissue could be seen. Towards the margin of the tumour this merged into dense and well-formed fibrous covering, resembling the structure of the dura mater. (Mixed cell sarcoma.)

Case 9.—L. G., aged 15 years, was admitted into the National Hospital, under the care of Dr. Beevor, on July 11, 1898. The family history was unimportant. Her health, up till the commencement of this illness, had always been good. In December, 1897, she began to vomit daily. In March, 1898, severe vertical headache first occurred and persisted, and she had diplopia. In June, 1898, she gradually became completely blind.

When admitted she was a healthy-looking child, and was able to get about. Intelligence good. No fits. Vision reduced to slight perception of strong light. Intense double optic neuritis becoming atrophic. Slight nerve deafness in right ear. The cranial nerves, limbs, trunk, sensation, and sphincters were normal. Gait normal. Knee, wrist, and elbow-jerks present and equal. On July 12 the knee-jerks could only be obtained with reinforcement. On August 1 the left knee-jerk was absent, as were the wrist and elbow-jerks. The right knee-jerk was a mere flicker. After this time she constantly complained of severe aching pain in the legs, from the knees down. The knee-jerks remained absent, but on a few occasions a trace of the right knee-jerk was obtained by reinforcement. On August 20 nystagmus and slight right facial weakness were noticed. The pains in the legs were troublesome, and came on in sharp twinges. No further localising signs appeared. She became comatose, and died on October 13.

Duration of symptoms ten months.

Description of tumour (plate viii.).—The convolutions were flattened and a small cystic projection was noticed on the inner border of the lower surface of the left temporo-sphenoidal lobe which proved on further examination to be the dilated horn of the lateral ventricle. After hardening in formalin, a horizontal section was made through the cerebral hemispheres so as to expose the capsule. The left hemisphere was seen to be occupied by a very large growth which extended from the tip of the occipital lobe to about 5 cm. from the tip of the frontal lobe. There was a hæmorrhage situated in the anterior portion of the growth.

The growth was yellowish grey in colour with a few punctiform hæmorrhages and measured 12 cm. by 6 cm. The right lateral ventricle was dilated. The examination of the other organs was negative.

Microscopical examination (plate viii.).—Bythe Marchi method, there was marked degeneration in both the postero-external and internal columns in the lumbar region. In the dorsal region the degeneration was most marked in the cornu commissural zone, while in the cervical region the degeneration was most marked in Burdach's column, and could be traced up to the funiculus gracilis and cuneatus, where it was evenly distributed throughout both bundles. The direct cerebellar tract showed no degeneration. The other tracts of the cord appeared normal though there were perhaps one or two degenerate fibres in one pyramid. No degeneration could be traced above the posterior column nuclei. The posterior nerve roots showed well-marked degeneration, and the degeneration was most marked in the eighth cervical and first dorsal.

By the Weigert-Pal method the cord appeared perfectly normal, and by the blue-black method the cells of the anterior horn appeared normal. Clarke's column and Lissauer's tract also appeared normal.

Nature of growth.—The growth was composed of irregular spindle-shaped cells, with small areas undergoing hyaline degeneration. Other areas had undergone a caseous degeneration and it was only at the outer margin of the tumour that the true nature of the growth was visible. (Mixed celled sarcoma.)

Case 10.—A. S., aged 12, was admitted on September 22, 1898, into the National Hospital, under the care of Dr. Buzzard.

Four months before admission, after an attack of influenza, she began to have frequent attacks of *petit mal.* She would suddenly turn pale and look strange, and say "I feel so funny." A fortnight later it was noticed that in some of the attacks the left arm twitched.

She was sent to the Meath Home for Epileptics, and when admitted there she was constantly falling asleep at all times of the day, though quite bright and natural when awake. It was then noticed that she did not use the left side of her face in talking so well as the right.

A month before admission her sight began to fail rapidly. A fortnight later paroxysms of vomiting began for the first time in her illness. She had had no headache until two days before admission. On admission she was obviously very ill. Her

mental state was intelligent. Speech and articulation were normal. She had frequent Jacksonian attacks, beginning sometimes in the left face, sometimes in the right face, sometimes in the right hand and sometimes in the left hand, remaining always confined to the side on which the attack commenced. She was quite conscious and able to speak during the attacks. Taste, smell and hearing were normal. There was intense optic neuritis showing a commencing subsidence into atrophy. There was absolutely no light perception. There was complete palsy of the right third and sixth nerves, and partial palsy of the left third and sixth nerves. There was very slight weakness of the left lower face and of the left arm. There was absolutely no weakness of the legs, and her gait was natural. There was no localised cranial pain or tenderness. Sensation was natural. The knee, wrist and elbow-jerks were absent and were never obtained up till her death. The plantar reflex on the right side gave a marked flexor response, that on the left side a vigorous extensor response. This condition of the plantar reflex persisted till the day before death, two months later, when she became deeply comatose, and the extensor response was present on both sides, but was much more marked on the left side.

A month after her admission she dragged the left foot slightly in walking, but after this the hemiplegia did not progress.

For six weeks after admission the clinical aspect of the case did not alter. She was up and about the ward. She did not suffer much pain, and her mental state was intelligent. She was never ataxic. A fortnight before death she gradually became drowsy and from that time slowly sank into coma.

Duration of symptoms six months.

Description of tumour (plate ix.).—The brain showed marked flattening of the convolutions, and the under surface of the right temporo-sphenoidal lobe was seen to be the seat of new growth. The lobe was swollen and enlarged transversely. On section the right temporo-sphenoidal lobe was found to be infiltrated by new growth and the descending horn of the lateral ventricle was filled by it. There was some dilatation of the lateral ventricle on the left side.

Microscopical examination (plate ix.).—By the Marchi method, sections of the cord taken from the cervical, dorsal and lumbar regions showed well-marked recent degeneration in both posterior columns, and it was almost as marked in the lumbar as in the cervical region. In the dorsal region the degeneration was most marked around the incoming roots, and in the lower and upper

cervical the degeneration was more marked in the columns of Burdach than in the column of Goll, though the degeneration was considerable in the latter column. The degeneration could be traced up to the level of the second cervical root (the cord was not available above this level). No degeneration could be found in the direct cerebellar tract. Some of the degenerate fibres that pass in by the posterior roots could be traced into the base of the posterior horns and ramifying there became lost among the cells of Clarke's column and also of the cells of the anterior horns. Posterior nerve roots: The posterior roots from the cervical, dorsal, and lumbar region all showed well-marked recent degeneration and this was slightly more marked in the cervical region than in either the dorsal or lumbar region. By the Weigert-Pal method sections taken at various levels in the cord show some degeneration in the posterior columns. In the upper cervical region the degeneration was more marked in the postero-external columns than in the postero-internal, but below this level the degeneration was evenly distributed. The degeneration as shown by this method, was not marked, but the columns are paler than the normal tracts.

By van Giesson's method the cells of the anterior horn appeared normal and there was no marked increase of the interstitial tissue in the posterior columns. Clarke's column appeared normal, but Lissauer's tract contained some degenerate fibres.

Nature of growth. — The growth is composed of cells of irregular shape lying in a matrix of fine fibrils. (Glioma.)

Case 11.—E. H., a coach-builder, aged 21 years, was admitted into the National Hospital under the care of Dr. Beevor on March 4, 1899. The family history was unimportant, and his health had been good until September, 1897, when headache, vomiting, diplopia, and failure of sight set in. Seven months later he had become completely blind. The headache and vomiting persisted, and in February, 1899, he became deaf in the left ear, and the left side of the face became paralysed, and he became very somnolent.

On admission he was somnolent, and his cerebration was slow. Speech normal. Taste and smell absent. Vision, no light perception. Complete post-neuritic atrophy of discs. Absolute nerve deafness in left ear. Divergent strabismus, the left eye looking down and out. Conjugate deviation to right was very feeble, and accompanied by slow long-range nystagmus. Marked paralysis of left face, both upper and lower. Electrical reactions of face

normal. Head retraction. Muscles of neck rigid. Slight weakness, tremor and incoordination of left arm. Gait normal, occasionally he staggered to the left. Sensation normal. Knee-jerks not well marked. Wrist and elbow-jerks absent. Double extensor response in plantars. Trephining for the relief of the intra-cranial pressure was resorted to and the lateral ventricle aspirated. Death from respiratory failure on March 27, 1899.

Duration of symptoms eighteen months.

Description of tumour (plate x.).—A large trephine hole existed over the left lateral lobe of the cerebellum. The skull was thinned almost to paper thickness on the right side of the middle fossa. The right temporo-sphenoidal lobe was noticed to be very adherent to the dura mater at its apex, and after removal of the brain from the cranial cavity a large encapsulated tumour measuring 7 by 7 cm. was seen to occupy the right middle fossa. It compressed the right temporo-sphenoidal lobe, and lay almost loose in the cavity so formed. It was firm, hard in consistence, and intimately adherent to the dura, from which it appeared to grow. All the other organs in the body were healthy.

Microscopical examination (plate x.).—By the Marchi method there was marked degeneration in the posterior columns from the sacral to the cervical region. In the lumbar region the degeneration was most marked in the postero-external columns, very little being seen in the postero-internal column. In the cervical region, although the degeneration was most marked in the postero-external columns, yet there was also considerable change in the postero-internal column. The degeneration could be traced up to the funiculi gracilis and cuneatus. In the region of the olive there was some degeneration in the ascending root of the fifth of one side. In the pons there was practically no degeneration, although some black dots could be seen in the fillet. No change could be found in the pyramidal system, and no degeneration existed in the direct cerebellar tract. There were, however, some degenerate fibres in Lissauer's tract.

Posterior nerve-roots: In the cervical region one or two degenerate fibres could be seen, but the degeneration was very slight when compared to that in the cord, and in the dorsal and lumbar roots no degeneration could be found. By the Weigert-Pal method, apart from appearing paler than normal, the posterior columns showed no change.

No abnormality could be detected either in the cells of the anterior horn or of Clarke's column by Nissl's method.

Nature of growth.—The growth consisted of spindle-cells with large nuclei and alveolar spaces. (Spindle-celled sarcoma.)

Case 12.—E. B., a clerk, aged 34 years, was admitted into the National Hospital under the care of Sir William Gowers on February 3, 1899. Seven weeks before admission, when in good health, he was suddenly seized in the night with headache, vomiting, and delirium. Severe pain in the right side of the head and vomiting persisted, and he had four or five slight fainting attacks, followed by mental peculiarity lasting some minutes. Fourteen days before admission his sight began to fail. On admission his mental state was facile and grandiose, not unlike that often seen in "general paralysis." Intense double optic neuritis. Weakness of both external recti and of the left side of face (cerebral type). Slight weakness and some tremor of left arm. Left wrist and elbow-jerk absent. Knee-jerks present L. > R. Plantar reflexes, flexor responses. The skull was opened for the relief of optic neuritis.

Subsequently severe left hemiplegia came on gradually, and he died of respiratory failure on April 17.

Duration of symptoms four months.

Description of tumour (plate xi.).—An opening in the skull was present over the right cerebellar region. There was marked flattening of the convolutions. Some adhesions to the dura mater were present over the right temporo-sphenoidal lobe. On examining the base of the brain the right temporo-sphenoidal lobe was found to be implicated by a tumour which protruded through the cortex in the hinder portion of that lobe. The edges of the tumour were hard and raised, and presented an irregular surface. The cerebellum on the same side had lost a considerable amount of its substance along the posterior margin owing to the operation. No other abnormality was noticed in the cerebrum or medulla.

On making a horizontal section through the brain a large tumour was seen occupying nearly the whole of the middle third of the brain on the right side. The margins of the tumour were ill-defined, and consisted of several layers, a pale yellowish central portion, a green translucent portion, and outside this the white tumour substance merged into the brain substance proper. The corpus striatum, the lenticular nucleus, and the internal capsule were all pushed forward by the tumour. The tumour measured 6 by 6 cm.

Microscopical examination (plate xi.).—(The examination of the head only having been allowed.) At the lowest point of the cord obtainable, viz., about the level of the first cervical root, there was marked degeneration in the posterior columns, Burdach being more affected than Goll. There was also degeneration in the decussating fibres of the pyramid.

In the medulla there was the same marked degeneration in the posterior columns, and there was also a good deal of diffuse staining, too general to be described in detail.

At the level of the olive there was a good deal of degeneration in the restiform body, and the substantia gelatinosa, and some degeneration in one pyramidal tract.

At the level of the posterior corpora quadrigemina there was degeneration in the decussation of the superior cerebellar peduncle, and the fillet on the one side contained a few degenerate fibres, and on the other side numerous degenerate fibres.

By the Weigert-Pal method the cord appeared normal. There was, however, a small, very pale area situated in the peripheral portion of the antero-lateral tract (probably a normal condition).

Nature of growth.—The tumour appeared to be an irregular-celled sarcoma. The cells varied greatly in size, and were set loosely apart. (Irregular-celled sarcoma.)

Case 13.—G. H., a coachman, aged 64 years, was admitted into the National Hospital under the care of Sir W. Gowers on July 7, 1899. His previous health had been good. Five weeks before admission he was noticed to be mentally strange. This continued till a fortnight later, when, on getting into bed, he fell down and immediately vomited. The next morning he was noticed to have weakness of the right face. After this right hemiplegia and aphasia slowly came on. He gradually sank into a comatose condition, and died six weeks after the onset of symptoms. Headache had not been complained of. Condition on admission :—Double optic neuritis. Complete right hemiplegia and aphasia, semi-comatose. The right knee-jerk was just obtainable, the other deep reflexes were absent.

Duration of symptoms six weeks.

Description of tumour (plate xii.).—The convolutions were somewhat flattened. Situated in the left temporo-sphenoidal lobe was a spot over which the vessels were dilated and on palpation, although the surface felt soft the subjacent tissue was hard and nodular. A similar patch existed in the situation of the left angular gyrus. The right lateral sinus was absent from its origin to the torcular herophili. On making a horizontal section through the brain, two tumours were seen, the one occupying the posterior part of the first and second temporo-sphenoidal convolutions of the left side, and extending deep into the cerebral substance, the other occupying nearly the whole of the anterior part of the temporo-sphenoidal lobe. This growth was entirely separate from the other tumour, and had undergone

some hyaline change. The lateral ventricles were not distended. The growth itself was hard and yellowish in colour, without distinct margins and obviously infiltrating the surrounding brain tissue. The one growth measured 5 by 3 cm. and the other 2 by 2 cm.

Microscopical examination (plate xii.).—By the Marchi method the lumbar and sacral region of the cord appeared perfectly normal. In the dorsal region there were a few degenerate fibres in the posterior columns, and in the upper cervical region there was a marked area of degeneration corresponding to about the fourth or fifth root on one side. This degeneration could be traced up to the funiculus cuneatus. The condition was present on both sides of the cord, but was far more marked on the one side than the other. There were also some degenerate fibres in the crossed pyramidal tract of the same side as that on which the greater amount of degeneration existed in the posterior columns.

The degeneration was slight when compared to some of the other cases. The posterior root ganglion examined was normal.

By the Weigert-Pal method the cord appeared perfectly normal. Lissauer's tract was normal.

By the Nissl method, although the cells in the anterior horn appear less numerous than normal, it could not be said that there was any pathological change. There were numerous corpora amylacea in the white matter of the spinal cord in all regions, but they were more numerous in the cervical than in other regions. These bodies were not visible by the Marchi or Weigert-Pal methods. Clarke's column was natural.

Nature of growth.—The growth was very irregular, hæmorrhage having occurred into it at various points, and at others it had undergone gelatinous degeneration. It was formed of a loose network with scattered nuclei. (Glioma.)

Case 14.—E. E., aged 50, was admitted to the National Hospital under the care of Dr. Beevor, on July 31, 1899.

One month before admission, her husband, on returning home one night, found her sitting in a chair, unable to move the right arm or leg, nothing was noticed in the face. She could speak correctly, but gradually lost sensible speech. She had had two or three attacks of vomiting since the onset of the illness, but had not complained of headache. She knew people about her and could indicate her wishes.

On admission, the patient could sit up in bed and could talk but not sensibly, about a week later she rather rapidly fell into a comatosed condition. The pupils were small, the right being

somewhat larger than the left, and neither reacted to light. There was slight squint due to weakness of the left internal rectus. There was slight neuritis in the right eye. Sensation was impaired all down the right side of the body and the corneal and conjunctival sensation was also diminished on the right side. The right arm and leg were absolutely flaccid. Reflexes: The arm-jerk on the right side was not obtained, but was present on the left. The right knee-jerk was not obtained, but the left was present. There was no ankle clonus. The plantar reflex on the right side gave an extensor response, and on the left stimulation on the inner side of the sole gave a flexor response while on the outer side an extensor response was obtained.

The patient gradually became more comatosed and died, and on the last day of life neither the knee-jerks nor the plantar reflexes were obtainable.

Description of tumour (plate xiii.).—There was flattening of the convolutions, especially in the region of the marginal and angular gyrus of the left side. On the right side there was no flattening or the convolutions which on the other hand appeared rather shrunken, and there was considerable compensatory œdema. On examining the base of the brain it was seen that the left crus was of a grey colour, and very considerably wider than the right. A section was made in a horizontal direction through the brain and a large tumour was seen to occupy the left hemisphere measuring 5·5 by 8 cm. The growth was situated in the posterior portion of the hemisphere, and extended forward to the hinder part of the left lenticular nucleus, and involved the left optic thalamus. The growth did not extend into the left lateral ventricle, but by its size had narrowed the limits of the ventricle. At no point did the growth reach the surface of the hemisphere. The growth appeared like a fleshy mass which had undergone yellow softening at its centre. The point of origin of the growth appeared to be about the optic thalamus. In a downward direction the growth extended into the left crus and tegmentum, but did not involve the corpora quadrigemina.

Microscopical Examination.—Marchi. No degeneration was found in the spinal cord either in the posterior or lateral columns. At the level of the olive only a very few degenerate fibres could be seen in the left pyramid and even in the pons where the growth actually invaded the left pyramid, degeneration could only be seen in the immediate neighbourhood of the tumour.

By the Weigert-Pal method no change could be detected in the spinal cord or the medulla; in the pons, however, there was

a large area of unstained tissue situated on the left side, and which involved nearly the whole of the left pyramid, fillet, and superior cerebellar peduncle.

By the Nissl method no change could be detected in the cells of the anterior horn.

Nature of growth.—The growth was composed of elongated nuclei arranged in a somewhat irregular manner in bundles which ran in various planes. There was very little alveolar structure, the growth being uniform throughout, and it was moderately vascular. (Spindle-celled sarcoma.)

Case 15.—W. W., aged 50, was admitted into the National Hospital, under the care of Dr. Tooth, on August 23, 1899.

Twelve months before admission he fell from a wall and struck the left side of his head, and ever since a week or two after this he had complained of headache at the back of his head. Six weeks after the accident he had a fit, in which he lost consciousness, fell, bit his tongue, and was convulsed all over. After this the fits recurred every fortnight. There was no warning at first, but his wife says that now he knows when a fit is coming on, and he has time to lie down.

During the fourteen days preceding admission the patient had vomited, but previous to this the patient had had no vomiting. His eyesight had been failing, and he had been "strange in his head"

On admission he was in a confused mental condition, but could answer questions, and used correct language. There was double optic neuritis with hæmorrhages. Both pupils reacted readily to light. There was slight right facial weakness, with distinct ptosis of the right eye-lid. Both arms and legs moved normally, but the limbs were flaccid.

Reflexes: Elbow and wrist-jerks were equal. The knee-jerk was present on the right side, and very difficult to obtain on the left. The plantar on the right side gave a flexor response while on the left it was sometimes absent, sometimes gave an extensor response. There was tenderness on pressure on the left side of the cranium above and also behind the ear.

The patient gradually became comatose, and died three days after admission.

Description of tumour (plate xiv.).—There was slight flattening of the convolutions and very marked bulging in the left temporo-sphenoidal region. The convolutions were broader than normal, and there was obviously a tumour infiltrating the left temporo-sphenoidal lobe; on section the growth was seen to occupy the

posterior and lower portion of the left temporo-sphenoidal lobe. The tumour had undergone cystic degeneration and the cyst was filled with green gelatinous material. The growth had no definite margins. The basal ganglia and the internal capsule appeared healthy and not affected by the growth. The growth measured 5 by 6 cm.

Microscopical examination.—Marchi's method (plate xiv.). The sacral, lumbar and dorsal cord appeared normal, and at the level of the fifth cervical there were two obviously degenerate roots; the one on the left side originated probably from the seventh cervical root, and the one on the right side from the sixth root.

The degenerated root on the left side was the more affected. This degeneration could be traced up into the funiculus cuneatus. No degeneration was present above the posterior column nuclei.

By the Weigert-Pal method the cord appeared perfectly normal, and by the Nissl method the cells of the anterior horn appeared normal.

Posterior root ganglion: The cells appeared perfectly normal, although there was considerable pigmentation.

Nature of growth.—The growth was composed of fibrous tissue with elongated nuclei, having an alveolar arrangement, the alveoli being filled up with nuclei of somewhat irregular size. In other portions of the tumour the alveolar arrangement was less marked. There was very little evidence of degeneration in the tumour, and at its margins where it infiltrated the brain it was extremely vascular. (Mixed-celled sarcoma.)

Case 16.—W. G. S., aged 7 years, was admitted into the National Hospital under the care of Dr. Tooth on January 6, 1899, complaining of vomiting and "tumbling about." There was a history of phthisis in the family.

The patient was quite well up till the age of 2 years, when it was noticed that he began to tumble about and to be clumsy with his hands. This had gradually increased till the time of admission. Since six months before admission he had vomited occasionally. No headache or fits had been noticed. On admission he was a particularly intelligent, healthy-looking child. Smell, taste and hearing acute. Vision $\frac{6}{24}$ and J 4 with each eye. Marked post-neuritic atrophy of both discs.

Marked spontaneous nystagmus, quick movement to right. Wide range nystagmus on deviation, more marked on deviation to right. Slight weakness of upper right face. Marked weakness of lower right face. Electrical reactions of face normal. Distinct bulging of skull over the left cerebellar region tender to

pressure. Intense jerky incoordination of all the limbs much more marked on the left side. Left limbs slightly less powerful than right. Reeling gait. He always fell straight backwards when standing up. Knee, wrist and elbow-jerks brisk and equal. Plantar reflexes were flexor responses.

The left side of the cerebellum was exposed by Mr. Horsley on February 2, 1899, and a cyst containing about an ounce of clear fluid evacuated, which appeared at one spot on the surface of the left lateral lobe of the cerebellum. As there was no obvious sign of growth in the cyst wall, after the evacuation of the cyst the wound was at once closed.

Three days after the operation the knee-jerks were absent. On the fourth day the right knee-jerk was obtained, but after this the knee-jerks were persistently absent. No other change in the physical signs took place. He died suddenly and unexpectedly eleven days after the operation.

Duration of symptoms three and a half years.

Description of growth (plate xv.).—There was an opening in the skull over the left lateral lobe of the cerebellum. The convolutions were not flattened, and the surface of the cerebrum appeared normal. The left lateral lobe of the cerebellum was adherent to the incision in the dura mater, but was easily separated from it, and on inspection presented very little abnormal. A hard tumour could, however, be felt, occupying the middle lobe and extending laterally more to the left than to the right. On section the tumour was found to involve the left corpus dentatum. It grew ventrally into the upper and left side of the pons, and extended upwards to a point just below the corpora quadrigemina. In a downward direction the growth seemed to involve the left middle and inferior cerebellar peduncle and the dorsal portion of the medulla on the left side.

Microscopical examination.— The cord appeared perfectly normal to all methods of examination; no degeneration could be shown by the Marchi method, even in the immediate neighbourhood of the tumour, though its limits are distinctly shown by the Weigert-Pal method.

Nature of growth.—The growth appeared to be formed of irregular-shaped cells; in some parts the cells were widely separated. There were numerous corpora amylacea in portions of the tumour. (Glioma.)

Case 17.—M. P., aged 47, was admitted to the National Hospital, under the care of Dr. Ferrier, on October 12, 1898.

For three to four years the patient had had a rushing noise

in the right ear. Eighteen months ago she found that she was deaf in the right ear. She had suffered from vertical and occipital headache for three years.

One year ago she had an attack in which the legs "went stiff" while walking; there was no loss of consciousness, and no twitching, and the stiffness was only momentary. Ever since this time she had been subject to occasional attacks of rigidity in all four limbs; she felt giddy, but did not fall down.

Three weeks ago her eyesight began to fail, and during the week before admission it had rapidly deteriorated. She had occasional attacks of complete blindness. She had had severe vertical headache for three years, occasionally spreading down the back of the neck. She had been very sick for a fortnight.

On admission she was a poorly nourished woman. Smell and taste were normal. She was totally deaf in the right ear. There was intense double optic neuritis. There was no weakness or ataxy of the trunk or limbs, and the gait was normal. The knee-jerks were normal and equal. There was no ankle clonus.

On November 30 Mr. Horsley removed the calvarium from the right occipital region, and on December 6 he opened the cerebellum, but was unable to find the tumour. The patient died on December 15.

Description of tumour (plate xvi.).—There was an opening in the right posterior fossa, due to the operation. The convolutions did not appear flattened. The base of the right lateral lobe of the cerebellum was broken up, as the result of the operation; projecting from the under-surface of the anterior part of this lobe of the cerebellum was a tumour measuring 3 by 5 cm., growing from the flocculus and pressing on the right side of the pons, so as to make it quite flat. The fifth nerve on this side was seen lying against the tumour, and was quite flat and tape-like.

Microscopical examination (plate xvi.).—By the Marchi method, there was marked degeneration in the funiculi gracilis and cuneatus, being more in the latter than the former. In the cervical region of the cord, the degeneration of the posterior columns was well marked, and affected the column of Burdach more than the column of Goll. Below the cervical region, the degeneration was much less marked, and was more scattered, but was present as low as the sacral region to a slight degree. There was no degeneration of any other tract in the cord. By the Weigert-Pal method, the cord appeared perfectly normal.

Nature of growth.—The growth was composed of elongated cells of uniform size and character, arranged in various planes. (Fibro-sarcoma.)

Case 18.—J. F., aged 5, was admitted to the National Hospital, under the care of Dr. Ferrier, on June 10, 1898.

Fourteen months before admission he had had whooping cough and broncho-pneumonia, and was in bed for ten weeks. Whilst in bed he had severe pain in the right side of the head, and his right arm was noticed to be weak. From this illness he recovered completely.

Five months before admission he had measles, and a month later he began to complain of headache. Six weeks before admission he was noticed to stagger, and complained of giddiness, but had neither headache nor vomiting.

On admission he was a well-nourished, intelligent boy. Double optic neuritis was present, but there was no weakness of the limbs nor ataxy while in bed. His gait was reeling and unsteady, and he tended to stamp the heels on the ground. He tended to fall when the eyes were shut. Reflexes: the knee-jerks were present and equal; there was no ankle clonus; the arm-jerks were equal. There was some prominence over the left side in the cerebellar region. On June 28, Mr. Horsley enucleated a tumour from the left lateral lobe of the cerebellum. The child developed symptoms of tubercular meningitis, wasted rapidly, and died on September 16.

Description of tumour.—There was a large opening in the left occipital region. The convolutions were flattened, and there was obvious tuberculous meningitis at the base. The left lateral lobe of the cerebellum was broken up and softened, and was adherent to the dura mater. The ventricles were much dilated. The spinal cord showed a marked degree of tuberculous meningitis, and this extended down to the lumbo-sacral region.

Microscopical examination of cervical region of the cord showed no degeneration either by the Marchi or Weigert-Pal methods.

Nature of growth.—Tuberculous.

Case 19.—L. S., aged 5 years, was admitted into the National Hospital, under the care of Sir William Gowers, on March 17, 1899, complaining of headache, loss of sight, and inability to stand or sit up. Seven months before admission, headache, vomiting, and inability to walk (staggering) had come on, and it was noticed that the head was constantly inclined to the right shoulder. Two months before admission she became unable to sit up, and she became gradually deaf in the left ear. For a month head retraction had been noticed.

On admission she was semi-comatose. Head retraction.

Hydrocephalic cry. Severe optic neuritis. Complete deafness of left ear. Pupils dilated. Nystagmus. General paresis and general coarse tremor on movement.

Knee-jerks present and equal. Plantar reflexes were extensor responses. On April 7, she developed measles, and the knee-jerks were noticed to be absent. She died on April 9, without further manifestation of symptoms.

Duration eight months.

Description of growth (plate xvii.).—The left lateral lobe of the cerebellum was enormously distended by a large cyst which appeared on the dorsal surface and compressed the right lobe across the middle line, the right lobe of the cerebellum appeared to be normal. After hardening in formalin a section through the cerebral hemispheres showed them to be perfectly normal and there was no distention of the ventricles. On section through the cerebellum the cyst was found to occupy almost the whole of the left lobe, only a thin layer of cerebellar substance covering the tumour. The cyst was filled with a green clear jelly-like substance, and measured 7 by 4 cm. in the antero-posterior and the lateral diameters respectively. The wall of the cyst measured about 2 mm. and was composed of two layers, the inner of which could be easily shelled out over its greater extent but was adherent and merged into the growth on the inner side of the lobe.

Microscopical examination (plate xvii.).—By the Marchi method there was marked degeneration of the posterior columns from the sacral to the cervical region, but the degeneration was more marked in the column of Burdach and the funiculus cuneatus than in the column of Goll and the funiculus gracilis. No degeneration could be seen above the posterior column nuclei. The posterior roots showed marked degeneration in the cervical region, less in the dorsal, and again more marked change in the lumbar region. By Weigert-Pal and Nissl methods the cord appeared normal. Clarke's column was normal and Lissauer's tract was unaffected.

Nature of growth.—The tumour showed characters that it was difficult to distinguish from brain tissue undergoing softening but it appeared to be a glioma with the cells ar apart. The cystic part of the tumour was found to consist of a meshwork of the finest fibres. (Glioma.)

Case 20.—W. G., aged 6 years, was admitted into the National Hospital under Dr. Tooth, on February 3, 1899, complaining of headache, sickness, and staggering. The family history

was unimportant and his health had been good previous to this illness. Three months before admission, headache and vomiting set in suddenly and persisted, and on several occasions he had attacks of trembling all over in his sleep. Six weeks later he began to walk badly and to tumble about.

On admission he was a healthy-looking child. He complained constantly of headache over the right eye. When sitting up the posture of the head was constantly as follows: right ear inclined to right shoulder, face turned to left, head bent forwards. Mental state intelligent. Special senses normal. Optic discs showed intense recent neuritis.

Marked nystagmus more intense on looking to right. Slight right lower facial weakness. Intense ataxy of left limbs, slight ataxy of right limbs. He walked unsteadily and reeled a great deal. When standing feet together he always fell straight backwards. Wrist and elbow-jerks present, knee-jerks sometimes brisk, sometimes difficult to elicit, even with reinforcement.

Mr. Horsley removed the calvarium and opened the dura mater over the left lateral lobe of the cerebellum. Some hours after the operation the patient became much worse, and the site of the operation was found to be bulging. On re-opening the flap a large tumour which had ruptured the left lobe of the cerebellum and escaped, was found lying free in the wound.

Description of tumour (plate xviii.).—There was marked flattening of the convolutions and dilatation of the ventricles. The left lateral lobe of the cerebellum was almost completely destroyed, being converted into a thin walled cavity from which a tumour measuring 7 cm. by 3·5 cm. had escaped. The cavity in the left lateral lobe was in direct communication with the fourth ventricle and extended upwards compressing the medulla and pons and dilating the iter as far as the posterior corpora quadrigemina. At a section made between the medulla and pons the cavity measured 3 cm. by 1·5 cm., and above this point it gradually narrowed down. The pons is so compressed that at its junction with the medulla it measured only 8 mm. in diameter. The tumour which had occupied the above cavity was dark in colour, friable, and evidently contained much blood.

Microscopical examination (plate xviii.).—By the Marchi method the cord in the cervical region showed degeneration in the posterior columns and in the direct cerebellar tract of both sides. Further examination was impossible as the removal of the spinal cord was not allowed.

Nature of growth.—The growth was an extremely vascular small round-celled sarcoma.

Case 21.—M. B., a woman, aged 30 years, was admitted into the National Hospital, under the care of Sir W. Gowers, on April 24, 1899. Three years before admission, occipital and right frontal headache set in; this was followed by giddiness, staggering, and diplopia, and a year before admission she became slowly blind, and also completely deaf in the left ear.

On admission there was no mental defect. Consecutive optic atrophy. Complete nerve deafness in left ear. Nystagmus most marked on deviation to left. Slight weakness of the whole left face. When standing the head always assumed the following position: the left ear was inclined to the left shoulder, and the face was turned slightly to the right. There was distinct bulging of the calvarium over the region of the left lateral cerebellar lobe. She fell to the left when standing. There was marked incoordination in the left limbs. She complained of severe pains in the legs, as if she had been walking too far. There was marked hypotonia of the muscles of the legs and a slighter hypotonia of the arm muscles. The knee-jerks were present and equal. Plantar reflexes were flexor responses.

The tumour was removed in part from the left lateral lobe of the cerebellum on June 20.

She died on July 20.

Duration of symptoms three years and three months.

Description of tumour (plate xix.).—There was an opening through the skull in the left occipital region. The surface and convolutions of the brain appeared normal; nothing abnormal was noted on the base of the cerebrum; the left posterior fossa was found to be occupied by a firm and hard tumour, adherent to the petrous bone, but not growing from it; the whole of the left lateral lobe was occupied by this tumour, and it pressed upon the left side of the pons, displacing it to the right and making a depression on its surface. An incision was made right through the cerebellum, and the tumour was seen to be infiltrated with blood, probably as the result of the operation. The tumour, on section, was hard and firm, and evidently very fibrous, although at places of a pale yellow colour, suggesting that it had undergone caseation. The lateral ventricles, as well as the third and fourth and iter, were greatly dilated.

Microscopical examination.—By the Marchi method, no degeneration could be seen, either in the posterior or other columns of the cord. In the dorsal portion of the medulla there was some degeneration in the restiform bodies and reticular formation, but this does not pass down the cord. The posterior root

ganglion appears normal, the cells being somewhat pigmented. By the Weigert-Pal and Nissl methods the cord appeared normal. Lissauer's tract and Clarke's column were also natural.

Nature of growth.—The growth appeared composed of elongated cells, arranged in bundles and running in different planes. The nuclei were very irregular in size, some being large and elongated and others small and round; the tumour was not unlike a fibroid tumour of the uterus. (A fibro-sarcoma.)

Case 22.—H. C., aged 7 years, was admitted into the National Hospital, under the care of Dr. Hughlings Jackson, on March 13, 1899. There was a family history of phthisis. His illness began a month before admission, with occipital headache and vomiting. A fortnight later he began to stagger in his walking.

On admission he was mentally very stupid, and presented the following signs:—Double optic neuritis. Nystagmus chiefly to right. Slight right facial weakness. Inco-ordination in all four limbs much more marked on right side. Gait staggering, leaning forward, and lurching to either side. No weakness or sensory change. Knee, wrist, and elbow-jerks R. > L., and subnormal; sometimes absent. Plantars flexor responses. He died on June 1, 1899.

Duration of symptoms four months.

Description of tumour (plate xx.).—The convolutions were flattened, but otherwise normal, and the surface of the cerebellum appeared natural. On section, however, a large tumour was seen situated exactly in the middle line, being composed of soft pulpy substance, limited by a sharp line from the cerebellar substance. The growth protruded into the fourth ventricle, and blocked it up. The growth measured 5 cm. in diameter, and was spherical in shape. Both lateral ventricles were considerably dilated.

Microscopical examination (plate xx.).—By the Marchi method, there was no degeneration in the sacral region of the cord; a little scattered degeneration was present in the posterior columns in the lumbar region, becoming more marked in the dorsal region and in the cervical, being fairly well marked in the postero-external columns, and slight in the postero-internal columns. There were a few scattered degenerate fibres in the pyramidal tracts of both sides. Lissauer's tract appeared normal. The cord appeared perfectly normal by the Weigert-Pal method, and the cells of the anterior horn also appeared normal by the Nissl method.

Nature of growth.—The tumour was composed of large nuclei, lying in what appears to be a nearly homogenous matrix;

in some parts of the tumour these nuclei were so densely packed together that hardly any matrix existed, while in other parts they were widely separated. The nuclei were very large and irregular both in size and shape. In other parts of the growth, the arrangement was more alveolar, with bands of fine fibrous tissue forming the alveoli. (Alveolar sarcoma.)

Case 23.—W. R., aged 30, a gardener, was admitted into the National Hospital, under the care of Sir William Gowers, on September 12, 1898. His illness began in January, 1898, with headache and vomiting. Since February, 1898, he had a constant feeling of unsteadiness when on his legs. In August, 1898, his sight began to fail rapidly and he staggered and fell repeatedly when walking.

On admission he was mentally very dull. There was intense optic neuritis going on to atrophy. He could just count fingers at six inches. No deafness. Marked nystagmus. Marked weakness of left face (upper and lower). Electrical reactions of face normal. Slight weakness of right arm and leg. He leant and fell to the right constantly when standing, and lunged to the right when walking. Sensations normal. Wrist, elbow, and knee-jerks subnormal. R. > L. Plantar reflexes were flexor responses.

During the next three months he became quite blind. Marked ataxy of the right limbs developed. The left facial palsy became more marked, no electrical alteration accompanying the weakness. The right hemiparesis of limbs became more marked. The deep reflexes remained. R. > L. and subnormal. The right knee-jerk was sometimes absent. Head retraction was noticed on several occasions. The plantar reflexes were always flexor responses.

He died on July 19, 1899.

Duration of symptoms nineteen months.

Description of tumour (plate xxi.).—On the under and external surface of the cerebellum is seen a dark, rounded tumour, hardly raised above the surface, with several large vessels running over its surface; on making a section through the cerebellum, a cyst, measuring 3·5 by 4 cm., is seen occupying the whole of the central portion of the right lateral lobe. The cyst is thin and easily separated from the surrounding brain substance, except at its outer side, where it forms part of the growth. There are several other smaller cysts filled with gelatinous material. In the left cerebellar lobe another growth could be seen occupying the central portion of the lobe; it was hard and firm, and had no

distinct margin separating it from the normal brain tissue that surrounds it.

Microscopical examination (plate xxi.).—By the Marchi method there was slight degeneration in the posterior columns in the sacral and lumbar regions, and in the dorsal it was still slight. It increased somewhat in the lower cervical region, where it was almost limited to the postero-external columns, and it was traceable to the funiculus cuneatus, the funiculus gracilis escaping altogether. By the Weigert-Pal method the cord appeared perfectly normal; the tract of Lissauer was also normal. By the Nissl method the cells of the anterior horn and of Clarke's column appeared normal.

Nature of growth.—The tumour was extremely vascular; there being large blood spaces surrounded by irregularly-formed fibrous tissue; in other spaces, where there was no blood, a fine meshwork existed, with scattered cells of a gliomatous appearance. In another portion of the tumour it appeared like cartilage, but the cell spaces were very irregularly formed. (Glio-sarcoma).

Case 24.—E., aged 6, was admitted to University College Hospital, under the care of Dr. Risien Russell, on September 21, 1898, with the history that, except for measles two to three years ago, she had been perfectly well till six to seven months before admission, when she had an attack of vomiting. A second attack occurred about three weeks later, with headache, and the attacks recurred at intervals of about a week ever since. She has been losing flesh rapidly of late. The sight has been failing for the last three to four days.

On admission the child was well nourished, the head was large, and the child was intelligent. The eyesight was defective, and optic neuritis was present in both eyes. There was no nystagmus, no facial weakness, and the hearing was equal on the two sides. No loss of power in the limbs could be found, but there was slight ataxy of the right arm. The gait was unsteady, and there was slight incoordination in the movements of the right leg. The head was in the right cerebellar attitude, *i.e.*, right ear inclined to right shoulder and face turned to left. The knee-jerks could not be obtained, and the wrist and triceps-jerks were also absent. The child died in October.

Description of tumour (plate xxii.).—The convolutions were flattened, and the lateral ventricles dilated. A large tumour was found to occupy the left lateral lobe of the cerebellum, and extended across the middle line. The tumour was of uniform consistence, with a fairly sharply-defined margin.

Microscopical examination of the cord (plate xxii.).—By the Marchi method there was some scattered degeneration in the lumbar region of the cord, limited to the postero-external columns. In the dorsal region the degenerated fibres seemed to have accumulated in the ventral portion of the posterior internal column. In the lower cervical region there is again more marked degeneration in the postero-external columns, and this can be traced up through the cervical region into the funiculus cuneatus. There were a few degenerate fibres in the funiculus gracilis. All the other regions of the cord appeared perfectly normal. By the Weigert-Pal method the lumbar and dorsal regions appeared perfectly normal. In the cervical region a small area, lying between the postero-internal and external columns, was paler than the other portions of the posterior columns, and seemed to contain fewer fibres. This pale area was symmetrical on the two sides.

Nature of growth.—The tumour was composed of cells lying in a meshwork of fine connective tissue; the growth appeared quite uniform in structure, and had undergone very little degenerative change. (Glioma.)

Case 25.—M. C., a nursemaid, aged 24 years, was admitted into the National Hospital, under Dr. Colman, on September 2, 1898, complaining of headache, sickness, and dimness of vision. Her father died of consumption, otherwise her family history was unimportant. She had always been strong and healthy till nine months before admission. Her sister stated that for five years she had been subject to neuralgic pain in the left temple, and that during this time she had attacks of dimness of vision, lasting a few minutes at a time. During the year preceding admission these attacks had been much more frequent. In January, 1898, she noticed dimness of vision at night. A month later severe headache and vomiting occurred and persisted. The headache, vomiting, and dimness of sight became steadily worse until a fortnight before admission, when the headache ceased. She had noticed some unsteadiness in standing for three months.

On admission she was a healthy-looking girl of good intelligence. She was evidently very ill, and was constantly vomiting. There was intense double optic neuritis, and vision was reduced to form-perception. Marked spontaneous nystagmus. She walked unsteadily, but on account of her general physical condition and the vomiting that the least movement brought on, no importance could be attached to this symptom. There was very marked muscular hypertonia of the limbs. The knee, wrist, and elbow-

jerks were absent, and were never obtained; otherwise, the special senses, cranial nerves, motion, sensation, and the sphincters were normal.

On September 17 she became delirious, and died suddenly six hours later. No fresh symptom had developed.

Duration of symptoms eight months.

Description of tumour (plate xxiii.).—The convolutions were not flattened, and the brain, as a whole, appeared normal in size and consistence. After hardening, a horizontal section was made through the two cerebral hemispheres, and an exceedingly vascular tumour was seen to occupy the posterior horn of the left lateral ventricle growing apparently from the choroid plexus. There was obvious hæmorrhage into the tumour, so that in colour it resembled hæmorrhage rather than growth. Its greatest length was in the antero-posterior diameter, being 5 cm., while its greatest width was about 3 cm. There was some blood-clot in the anterior horn of the left ventricle.

Microscopical examination (plate xxiii.).—By the Marchi method there was some degeneration in the posterior column of the cord, most marked in the mid-dorsal region, where the degenerate roots can be seen entering the cord, and this degeneration was traceable up the cord, becoming more diffuse in the cervical region. The degeneration was only of moderate degree, and at no level could be said to be marked. There were a few degenerate fibres in the lumbar and sacral region of the cord. By the Weigert-Pal method the cord appeared perfectly normal.

Nature of growth.—The growth was extremely vascular, composed of cells of irregular size and shape, lying widely separated in a matrix which stained badly, and in which degeneration had taken place. There were numerous masses of brown pigment, probably altered blood clot. (Glio-sarcoma.)

Case 26.—D. C., a clerk, aged 27 years, was admitted into the National Hospital, under the care of Dr. Bastian, on February 14, 1898. In February, 1897, he constantly fell asleep over his work, and from this time left hemiplegia slowly developed. In September, 1897, he had headache for the first time, and his sight began to fail. Up to the time of his admission he had had no fits and no vomiting.

On admission his mental state was intelligent. He frequently dropped off to sleep at his meals. Double optic neuritis. Rigid left hemiplegia. No hemianopia. No hemianæsthesia. Knee, wrist and elbow-jerks L. > R. Left foot clonus.

On May 10 he was semi-comatose, and vomited frequently. Left hemianæsthesia was present. He was absolutely blind.

On August 22 left-sided fits, without loss of consciousness, occurred and recurred at intervals for some months. The left hemiplegia became absolute.

In October right hemiplegia slowly came on, and after this right-sided convulsions, without loss of consciousness, occurred. Double foot clonus and double extensor response developed. Death occurred on February 8, 1899, from respiratory failure.

Duration of symptoms two years.

Description of tumour (plate xxiv.).—There was marked flattening of the convolutions on both sides, with bulging on the right side. On section through the brain, a tumour, measuring 4·5 cm. by 4·5 cm., was seen to occupy the right optic thalamus, and extended down into the right crus. The tumour was of yellowish colour, and built up of concentric laminæ; it was extremely hard, and showed no tendency to soften. The lateral ventricles were dilated. There was an old focus of tubercle at the apex of the left lung.

Microscopical examination (plate xxiv.).—By the Marchi method degeneration in the pyramidal tract of the right hemisphere could could be traced through the pons into the medulla and down to the sacral region of the cord. Sections through the middle of the pons showed degeneration in the cerebellar peduncle of the same side, and also, to a lesser extent, in the fillet. At the level of the olive there were some scattered black dots in the raphe, and definite degeneration in the fibres of the twelfth nerve. The fillet degeneration could be traced down across the raphe into the nuclei gracilis and cuneatus of both sides, the opposite one being, however, most affected. There was some degeneration in the direct cerebellar tract on both sides. Below the decussation of the pyramids there was very little degeneration in the posterior columns, and that was limited to the cervical region. Some degeneration could be seen in the posterior longitudinal bundle of the same side as the lesion. By the Weigert-Pal method there was marked degeneration of the pyramid, both direct and crossed, but the posterior columns were natural. There was marked degeneration in the fillet of the same side, but both posterior longitudinal bundles and superior cerebellar peduncles appeared normal. By the blue-black method the cells of the anterior horn and of Clarke's column appeared normal. The right and left median and anterior crural nerves were examined, and the left median showed most extensive change, not only by the Marchi but also by the Weigert-Pal method; the other nerves appeared normal.

Nature of growth.—On section, the growth showed a degenerate centre surrounded by a large number of small round cells, among which are many well-formed giant-cells; so marked was this appearance that the tuberculous origin of the tumour could not be doubted; no tubercle bacilli could, however, be found. (Tuberculous tumour.)

Case 27.—H. F., aged 18 months, was admitted into the National Hospital, under the care of Dr. Buzzard, on May 9, 1899. Eight weeks before admission, strabismus and head retraction were noticed. A few days later the sight was noticed to fail. At the end of a fortnight paralysis "all over" came on, and the child lost its speech. On admission he was semi-comatose, with convergent strabismus and complete ophthalmoplegia in right eye. Nystagmus in left eye. Neither pupil reacted to light. Optic discs normal. General paresis more marked on the left side. Head retraction. Slight hydrocephalus. The knee-jerks were absent, but on striking the left patella tendon, an adductor-jerk was obtained on the opposite side. He died on May 19.

Duration of symptoms nine weeks.

Description of tumour (plate xxv.).—There was marked flattening of the convolutions, otherwise the brain appeared normal. After hardening in formalin, the brain was cut in horizontal section. Both ventricles were enormously distended. A tumour was seen occupying the right optic thalamus; it extended into and involved the corpora quadrigemina, and passed over to the optic thalamus of the opposite side. It extended downwards to the commencement of the pons, and ventrally to the substantia nigra, but did not involve the crura. The growth was an infiltrating one, without definite margins, and towards the centre had a yellow granular appearance, but was hard and not caseous. Hæmorrhage had taken place into the tumour.

Microscopical examination (plate xxv.).—By the Marchi method there was very marked degeneration in the sacral region of the cord, leaving the septo-marginal tract almost unaffected, the degenerate fibres could be traced into the base of the posterior horns, and some also into the anterior horns. In the upper lumbar region the degeneration was fairly evenly distributed, but left the dorsal and median portion of the cord less affected; in the mid-dorsal region the column of Goll was more affected than that of Burdach. In the lower cervical region the entering roots were markedly degenerate, and the column of Burdach was markedly affected. The degeneration could be traced up to the nuclei

gracilis and cuneatus. The direct cerebellar tract was unaffected, and the tract of Lissauer also escaped. In the medulla there was some degeneration in the fillet and in the remaining portions of the funiculi gracilis and cuneatus. In the pons the degeneration was again more extensive, and involved the posterior longitudinal bundle, the fillet, and the superior cerebellar peduncles on both sides. The posterior root ganglia, so far as the cells were concerned, appeared normal both to the Marchi and Nissl methods. Posterior nerve-roots appeared perfectly normal; no degeneration could be seen in them. By the Weigert-Pal method the cord, medulla, and pons appeared perfectly normal; Lissauer's tract was normal. By the Nissl method the cells, both of the anterior horn and of Clarke's column, appeared natural.

Nature of growth.—The growth was very vascular at its margins, and was formed, for the most part, of large round cells of uniform size, with distinct nuclei, there being comparatively little fibrous tissue; but at certain points, generally in the neighbourhood of vessels, there was extensive infiltration, with small round cells (sarcoma).

Case 28.—A. A., aged 7, was admitted into the Hospital for Sick Children, under the care of Dr. Lees, with the following history:—She had been quite well till eleven days previously; she then complained of pain in the back of her head, held her neck stiffly, and vomited frequently.

On admission she was drowsy, but would answer questions; there was slight facial weakness on the left side; loss of sensation of the left cornea and conjunctiva, and paralysis of the left external rectus. There was double optic neuritis. The knee-jerks were present. The child died the day after admission.

Description of tumour (plate xxvi.).—The convolutions were flattened, and there was considerable dilatation of both the lateral ventricles as well as the third and fourth. The pons appeared very much enlarged, and the left side larger than the right. On section, a firm, white, infiltrating growth was found to occupy the left side and extended across the mid-line into the right side. The other organs of the body were normal.

Microscopical examination.—No degeneration could be detected in the spinal cord either by the Marchi or the Weigert-Pal methods, and the cells appeared normal by the Nissl method. Section of the pons stained by the Weigert-Pal method show a large infiltrating tumour separating the fibres of the pons, but apparently not destroying them.

Nature of tumour.—The growth was composed of a meshwork

of fine fibres, among which were scattered irregularly nuclei of uniform size, mostly angular in shape, and from some it would seem that fine prolongations can be traced into the meshwork. The growth is quite uniform in structure. (Glioma.)

Case 29.—E. J., aged 42, was admitted into the National Hospital, under the care of Dr. Beevor, on August 24, 1899.

Fourteen days before admission the patient began to have giddiness and severe neuralgic headaches, but previously to the onset of these she had been in particularly good health. The pain was referred to the frontal region of the skull. The headache increased, and twelve days after the onset she was found by her daughter in a dazed condition, complaining of intense pain in the head. She had an attack of vomiting. About 5 o'clock on the same day she was found lying on the bed in a semi-comatose condition. The dejecta were passed under her involuntarily; the coma deepened, and she was admitted into the hospital two days later.

On admission the patient was well nourished, and lay in bed with the legs drawn up, the arms flexed, and the head turned into the pillow, and all attempts to alter this position were resented. Respiration tended to assume the Cheyne-Stokes character; the right pupil was larger than the left, and did not react to light (previously dilated with atropine). The discs were hyperæmic, the edges blurred and streaky, and the veins swollen. No loss of power could be detected; the lower limbs tended to assume the flexed position, but were not rigid. Reflexes: the arm-jerks were present; the knee-jerks were present, but were not brisk; there was no ankle clonus, and the plantars gave a flexor response. No abdominal reflex was obtained. Urine 1024, no albumen or sugar.

Death occurred at 3 a.m. on the following day, respiration ceasing suddenly, followed after an interval of some minutes by heart failure.

Description of tumour (plate xxvii.).—There was marked flattening of the convolutions, but no tumour could be detected on the surface. On making an incision through the callosum, a cyst was seen lying below the anterior portion of the fornix, filling up the lateral communications of the lateral ventricles, and pressing down into the third ventricle, blocking up the foramen of Munroe. The cyst was somewhat pear-shaped. It was adherent to both choroid plexuses, and probably grew from the left. There were numerous small cysts in both plexuses. On cutting through the anterior portion of the fornix, the cyst, fornix, and choroid

plexuses could all be turned back together. There was dilatation of both ventricles, but not to an extreme degree. The basal ganglia were normal. On making an incision into the cyst, after hardening, it was found to consist of a thin wall, within which was a clear hyaline substance firmly set by the re-agents in which it had been hardened.

Microscopical examination.—The cord and medulla were examined by the Marchi, Weigert-Pal, and Nissl methods. No degeneration or change could be found.

Nature of Growth.—The cyst was composed of three layers, an outer formed of elongated cells with rounded nuclei, closely packed together, making a fibrous-looking coat; a middle layer composed of large epithelial cells, like the prickle cells of the rete mucosum of the skin; and an inner layer of structureless tissue, forming the greater part of the tumour. The transition of the prickle cells into the hyaline tissue could be traced. (Cyst of the choroid plexus.)

EPILEPTIC ATTACKS WITH A WARNING OF A CRUDE SENSATION OF SMELL AND WITH THE INTELLECTUAL AURA (DREAMY STATE) IN A PATIENT WHO HAD SYMPTOMS POINTING TO GROSS ORGANIC DISEASE OF THE RIGHT TEMPORO-SPHENOIDAL LOBE.

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THE patient, A. B., a member of the medical profession, was 51 years of age, married, and of excellent health until three and a half years ago. He then had a severe attack of septicæmia, during the course of which an abscess formed in his anterior chest-wall and required to be opened surgically. From that illness he recovered and remained well until October, 1898, when he had a fresh septic infection in his right hand, which required several incisions. At that time his temperature was over 105° on several days and he was occasionally delirious. Since his second attack of septicæmia his general health has never been quite so good. There was no history of specific disease nor of any exposure to it. He never had any head injury nor had he, at any time, suffered from otorrhœa.

Ever since his second attack of septicæmia the patient had been subject to occasional attacks of excitement, without sufficient apparent cause. He used to come home from his rounds in a state of excitement. His general manner became different; his memory also became impaired, so that on several occasions he forgot important appointments. On one occasion he called to enquire for a patient who had long been dead.

About eight weeks before he came under our observation he began to have occasional attacks of nausea and what he called

"giddiness," with frontal headache. In these attacks there was a strong sensation of smell, which he described as being "like camphor or ether." This smell was succeeded by what is commonly called an intellectual aura (dreamy state), in which—to use his own words—he "felt as if he were saying, doing, and looking at things which he had experienced before." He also stated that surrounding people seemed to have strange expressions on their faces, and that "people and things seemed to be far away" (he meant in physical distance). The average duration of each attack was about a minute; the patient's face, according to the account of a competent eye-witness of several seizures, one of which was very carefully observed throughout, was pale and his expression was that of one "facing an expected horror," but he was not apparently unconscious. No twitchings of face or limbs were ever observed. There was no vomiting either during or between the attacks.

After four weeks of such attacks, the crude sensation of smell ceased to occur, but there were numerous attacks, sometimes three in a day, of a slightly different type, in which he had nausea and vertigo. External objects did not appear to move, but the patient himself felt giddy, and he had a sense of fear and of impending death. Occasionally, during such an attack, he became momentarily unconscious and fell backwards. His face was always pale during an attack, and his pupils were dilated.

He never had diplopia, though six weeks after the commencement of his "attacks," when lighting a cigarette one day, he applied the lighted match to the cigarette-holder, apparently owing to some mental confusion.

His headache gradually became almost constant, the pain being both frontal and occipital, but within a week or so before he came under our observation the headache became less severe, and he seemed rather drowsy. Three weeks before, his left foot was noticed to drag a little in walking, but he could walk fairly long distances until he took to his bed, a week before we saw him.

His condition, on examination on September 11, 1899, was as follows:—He is a very intelligent man, able to answer clearly all questions put to him, though he seems somewhat drowsy; speech and articulation are normal. He can smell asafœtida and cloves in both nostrils, and names them. He can taste sugar and salt equally on both sides of the tongue, back and front. He hears a watch better in the right ear than in the left, and Mr. Ballance, on applying Weber's test, finds that the tuning-fork on the vertex

is heard louder on the right side, all wax being previously removed from the right ear. The visual fields are normal on rough testing. There is no hemianopia on careful testing with small objects moved at the periphery of the visual fields, nor is there any obvious defective sight.

On ophthalmoscopic examination there is found well-marked neuritis of the right eye, with + 2 D of swelling there are no hæmorrhages; the left optic disc is red but not swollen. The pupils are equal in size, and react normally to light and during accommodation. The external ocular movements are normal. There is no squint, no diplopia and no nystagmus.

There is slight but distinct weakness of the left facial muscles, more marked in the lower than the upper part of the face. The left angle of the mouth is less energetically retracted and the left naso-labial fold shallower than on the right side. The left eye is less tightly "screwed up" by the orbicularis palpebrarum than on the right side when he is told to close both eyes as strongly as he can.

The palate rises normally on phonation. The tongue comes out straight on protrusion, but can be pushed better into the left cheek than into the right.

There is no anæsthesia anywhere on the face, trunk, or limbs to the lightest touches or pricks, but when the fingers of the left hand are touched and the patient is asked to point with his other hand to the exact point touched, his eyes being closed, he is several inches wide of the mark in every instance. There is no constant relation between the point touched and the position to which he refers the sensation.

The sense of position on passive movement is normal at all joints of the upper and lower extremities.

As to motor power, the patient can move all parts of the head, trunk and limbs as he lies in bed, but the left grasp is much weaker than the right. (L. = 30, R. = 63, with dynamometer.) There is no ataxia of the limbs and no tremor on voluntary movements.

The patient cannot stand alone; when supported he tends to fall backwards and slightly to the left side; in walking he scrapes the left foot very slightly on the ground.

The knee-jerks are normal and equal. There is no ankle clonus. The right plantar reflex constantly shows flexion of the hallux. The left side is sometimes flexion, at other times extension of the hallux. The sphincters are normal.

The lungs and heart are normal on physical examination;

the pulse is 70, regular, and of moderate tension. There is a scar of an old abscess on the front of the chest. The abdomen and urine are normal. The temperature is normal.

To-day, for the first time, he complains of unilateral headache, and indicates the right temporal region as the seat of pain. There is no tenderness on pressure or percussion over any region of the skull.

September 12.—During last night the respiration was of Cheyne-Stokes type, and the patient passed urine and fæces unconsciously. To-day he feels better and talks with perfect intelligence. The headache is less severe. His appetite is extremely good, and he can smoke cigarettes. The left facial weakness is more evident than yesterday. The pulse is 72, respiration 20 per minute. The knee-jerks are very faint, and can only be elicited by Jendrassik's method.

September 13.—During the night patient's temperature rose to over 100° F. He rapidly became comatose, and was observed to have occasional clonic spasms, confined to the left arm and hand. Death occurred at 1 a.m. from failure of respiration. The temperature at the time of death was not taken.

Unfortunately no autopsy could be obtained.

Summary.—To sum up the history of the case: the patient had gradually increasing mental dulness, followed nine months later by attacks of headache and nausea with a sensation of smell and a "dreamy state." Later the attacks changed in type. The paroxysmal smell ceased to occur, but there was occasional momentary unconsciousness. A few days before his death slight left hemiplegia developed, most marked in the face, without hemianopia or hemi-anæsthesia, but with some "cortical anæsthesia" of the left hand. There was some impairment of hearing in the left ear and optic neuritis in the right eye. Two days before death the headache became more localised and was referred to the right temporal region. The patient rapidly became worse and died from failure of respiration, slight jerking being observed in the left arm and hand just before death.

Remarks by Dr. Hughlings Jackson.

I saw A. B. with Sir William Gowers about September 9, 1899. We concluded that there was disease, probably tumour, of one temporo-sphenoidal lobe. Sir William,

from the right-sidedness of the optic neuritis, suggested before the setting-in of the left hemiplegia, that the right lobe was the one implicated. I saw A. B. again a day or two later, this time with Mr. Ballance and Dr. Purves Stewart. Mr. Victor Horsley also saw the patient. The question of operation was considered; it was decided not to operate.

It is very unfortunate that necropsy was not permitted, but the case is clinically a very important one, especially, I think, as exhibiting the association of a warning of a crude sensation of smell with the "dreamy state," and with the emotion fear, in slight epileptic seizures. The notes were taken by Dr. Purves Stewart. Dr. Stewart will remark on the question of localisation of the lesion, on the olfactory and paralytic symptoms, and on some other points.

I have published a case somewhat like that of A. B. (BRAIN, July, 1888, part xlii., p. 191), but unfortunately necropsy was not permitted; that case,[1] however, is one of great clinical interest. Dr. Stewart will mention other cases already published of epileptic fits with crude (warning) sensations of smell, cases completed by necropsies.

I have several times[2] written on the group of cases of epilepsy to which that of A. B. belongs; and have (*Lancet*, January 14, 1899), suggested for these cases the name of Uncinate Group of Fits; this was on the hypothesis that the discharge-lesions in these cases are made up of some cells, not of the uncinate gyrus alone, but of some cells of different parts of a region of which this gyrus is part—a very vague circumscription, I admit—the uncinate region. In cases of this group there is at the onset of the paroxysms a crude sensation of smell or one of taste, or there are movements of chewing, smacking of the lips, &c.,

[1] The title given to the report of the case is a short summary of its symptomatology: "Epileptic Attacks with crude Sensation Warnings, by Smells and the 'Epigastric' Sensation, 'Intellectual Aura,' or 'Dreamy State.' Double Optic Neuritis. Attacks of Left-sided Tremor. Apoplexy and left hemiplegia. No necropsy."

[2] *Med. Times and Gazette*, vol. i., 1879; BRAIN, July, 1880; BRAIN, July, 1888; BRAIN (with Dr. Beevor), Oct., 1889; BRAIN (with Dr. Colman), Winter Number, 1898; *Lancet*, January 14, 1899.

(sometimes there is spitting). In some cases of this group there is a warning by what is known as the epigastric sensation, a crude development of a systemic sensation; this warning sometimes occurs along with a crude sensation of smell or with the chewing, &c., movements. Different varieties of this group of cases depend, I suppose, on discharge-lesions of different parts of what I call the uncinate region. As will have been inferred, it is supposed that especially in this cortical region, not confined to this region, are the physical bases of some of the systemic sensations—the physical bases of those systemic sensations which, to speak very roughly, especially appertain to the digestive system.

Many of the symptoms of even slight paroxysms of uncinate fits depend, of course, on discharges wide-spread beyond, some far beyond, the uncinate region—depend on discharges *secondary* to the *primary* discharge, that of the discharge-lesion.

There is the dreamy state in the paroxysms of many, not of all, cases of the uncinate group of epileptic fits.

I hope soon to be able to write more fully on the uncinate group of epileptic fits. I shall then consider exceptions to the statements made in the preceding paragraph, and shall re-enquire particularly as to the relation in slight epileptic paroxysms of crude auditory sensations to the dreamy state.

A. B. had only slight epileptic attacks, *le petit mal*, and two degrees of them; in the slightest of his slight attacks he had crude sensations of smell and the dreamy state; in the severer of his slight attacks there was "unconsciousness" without the crude sensation and without the dreamy state.

The dreamy state in the uncinate group of epilepsies varies very much in different cases.[1] In A. B. it was the peculiar intellectual state which many healthy people or people slightly out of health have occasionally—Reminiscence.

[1] As I have said, the dreamy state does not occur in the paroxysms of every case of the uncinate group, and I now say, too, that the dreamy state may, according to statements of patients and their friends, occur without any one of the crude sensations I have mentioned in the text, and without the chewing, &c., movements.

It is quite evident that in some slight epileptic paroxysms with the dreamy state—in all of them, I believe—there is a kind of double consciousness—a "mental diplopia." In a quotation from Herpin (*vide infra*) will be found clear evidence of "mental diplopia" in several of the cases he mentions. I think, too, that there is probably some defect of consciousness in every paroxysm with the dreamy state; certainly there is in some of them. The psychical condition, therefore, is a very complex one. There is defect of consciousness, so far negatively. Positively, there is (1) the quasi-parasitical state of consciousness (dreamy state), and (2) there are remains of normal consciousness; and thus (1 and 2) there is double consciousness. To discover the physical conditions correlative with this complex psychical condition is a very difficult task. There was also an abnormal[1] emotional state in some at least, of A. B.'s severer slight attacks—fear, *and a sense of impending death;* in the slighter of his slight attacks there was an expression of "one facing an expected horror" (*see* Herpin, *Des Accès*, &c., p. 111, quoted at the end of these remarks). I have said, *Med. Times and Gazette*, March 1, 1879, that the epigastric sensation in epileptic paroxysms is often accompanied or quickly followed by the emotion fear. Sir William Gowers thinks that this association is very rare. A. B. had no epigastric sensation warning.

We must not, in this part of our investigation of epileptic paroxysms, limit enquiry to the symptom fear; we have to take heed of all departures from the patient's "ordinary state of feeling." And it must be well borne in mind that the departure in uncinate paroxysms is in some cases towards a more pleasurable, although more often towards a more disagreeable, state. Further, what is obviously equally significant, early in the course of a case of epilepsy the feeling in the slight paroxysms may be pleasurable, and, later, disagreeable—differences depending probably on the degree and rate of the discharge of the discharge-lesion.

The symptom at the onset of A. B.'s attacks—that

[1] I say abnormal because, of course, I do not mean the natural fear of the fit itself, but a fear which "comes by itself"—the *symptom fear*.

external objects seemed to be far away—deserves careful attention.

In, or before, or after some slight paroxysms of cases of the uncinate group, there are gastric or intestinal symptoms, or both.

To speak very roughly, much of the symptomatology of uncinate fits refers very especially to the digestive system; the crude sensations, smell, taste, and the epigastric sensation are, to speak with brutal disregard of psychological propriety, crude developments of "digestive sensations."[1] In what, for reference, I will call Herpin's "third smell case" (*vide infra*), there were in the patient's paroxysms crude sensation of smell, abnormal intellectual and emotional states, and gastric spasm. The dreamy state, an abnormal intellectual state, is not a "digestive symptom," nor is an abnormal emotional state. But the occurrence in healthy people of gastric and intestinal states, things physical, along with the psychical states, pleasure and pain, is known to everybody ("bowels of mercy," &c.). The occurrence of gastric and intestinal symptoms in some cases of uncinate fits with abnormal emotional states is obviously significant.

I take this opportunity of advising the younger medical neurologists to study carefully Herpin's writings on Epilepsy. I have long known his valuable work, *Du Pronostic et du Traitement Curatif de l'Epilepsie*, 1852, but I have only recently heard of his still more valuable work, *Des Accès Incomplets d'Epilepsie*, 1867. I give, from the latter work, a quotation which mentions one case of the association of an intellectual aura ("dreamy state") with a crude sensation warning of smell in epileptic paroxysms ("third smell case").

Among three hundred cases of epilepsy observed by Herpin there were three, the paroxysms of which began by a warning of smell. In the report of only one of these

[1] Although smell takes low rank as it serves as a "digestive sensation," it has very high rank in another way. It has been called the most suggestive of all the senses; and smells have remarkable power in calling up remembrances of past scenes. We must, however, for several reasons, not be hasty in concluding anything from this with regard to explanations of the nature of the association of crude sensations of smell with the dreamy state in slight epileptic paroxysms.

three (I called it Herpin's "third smell case") is what I take to be a "dreamy state" ("tristes souvenirs"—"un trouble intellectuel") mentioned; the patient's emotional state in her paroxysms, from the description Herpin gives in the words I italicise, was similar to that A. B. had ("sense of fear and of impending death") in those of his paroxysms in which he lost consciousness. Herpin writes, *Des Accès Incomplets d'Epilepsie*, pp. 115 and 116, 1867 :—

"L'un de trois malades à début par de fausses perceptions d'odeurs perdait si vite connaissance qu'il ne pouvait que rarement avertir. Un autre sentait l'odeur descendre dans la gorge; alors toute perception se perdait. La troisième éprouvait une sensation qui de la gorge descendait dans l'estomac où elle se convertissait en inquiétude et en tortillements; pendant ce spasme, de tristes souvenirs assiégeaient la patiente; *il lui semblait qu'elle allait mourir;* aussi, quoique l'odeur fût agréable, l'impression qu'elle en éprouvait [était] des *plus pénibles*, mais bientôt elle perdait ses sens.

"Dans les débuts par l'organe olfactif, l'abolition de la conscience suivait donc le plus souvent le prélude; dans un cas, celui-ci était suivi d'un *spasme gastrique précédant un trouble* intellectuel pendant lequel la connaissance se perdait."

In the following quotation from p. 140 *op. cit.*, Herpin mentions a case so similar to the third case mentioned in the foregoing quotation from pp. 115 and 116, that, I think it is the same case—that there is, in one of the two reports (pp. 115 and 140), some mistake in the words which indicate sex.

"De trois patients à début par de fausses perceptions d'odeur, deux n'avaient pas d'accidents moindres que les vertiges. Le troisième avait quelquefois de simples préludes; la mauvaise odeur ne s'étendait pas au de là des fosses nasales (tandis que dans le vertige la sensation descendait dans la gorge); bientôt tout était fini; il n'y avait pas trace de trouble intellectuel; il appelait ces accidents légers 'ses *mauvaises odeurs.*'" In this third case (supposing it to be the same as the third case Herpin mentioned, pp. 115 and 116), the paroxysms were sometimes very slight indeed, simple

preludes; there were then crude sensations of smell without the "trouble intellectuel"; in somewhat stronger slight fits there was the "trouble intellectuel" also—differences due to differences in the degree of "intensity" of the primary discharge, that of the discharge lesion, and consequent differences in the range of the induced (secondary) discharges of the normal nervous elements with which the fulminant cells of the discharge lesion were connected.

I have several times drawn attention to the importance of taking careful note of chewing, &c., movements in epileptic paroxysms; they occur in one variety of uncinate fits, and sometimes there is, as in the other varieties, the dreamy state with them, and sometimes, as in the other varieties, there are elaborate actions during so-called unconsciousness after such seizures. There were no such movements in A. B.'s paroxysms.[1] I published (with Dr. Colman) a case in which there were such movements in the epileptic paroxysms of a patient who had the dreamy state (case of Z., BRAIN, Winter Number, 1898), small focus of, or relics of, softening in the left uncinate gyrus. As I have several times mentioned, I consider in accord with certain of Ferrier's inferences from experiments on the monkey, that these movements signify—are indirect consequences of —epileptic discharges of cortical gustatory elements. I quoted an account of an epileptic paroxysm with these

[1] Most neurologists will have read the report of a very remarkable case published by Dr. Mesnet (*L'Union Medicale*, Juillet 21 et 23, 1874), a case of what he calls "Pathological Somnambulism." I draw attention to the words of Dr. Mesnet, which I have italicised in the subjoined quotation.

He writes of his patient: Sa démarche est facile, son attitude calme, sa physionomie paisible; il a les yeux largement ouverts, la pupille dilatée; le front et les sourcils contracturés, avec un mouvement incessant de nystagmus accusant un état de malaise, de souffrance vers le tête; *et un mâchonnement continu.*" There is no mention of a dreamy state, and it appears that the "mâchonnement" was not at the onset of the paroxysms only.

This case is remarked on by the late Professor Huxley in an article "Animal Automatism" ("Collected Essays," vol. i.). The case has also been remarked on by Azam (Tuke's "Dict. of Psych. Med.," article, "Double Consciousness"), and by the late Hack Tuke, in his work on Somnambulism. Huxley alone mentions the "chewing motion of the jaws."

Without expressing any opinion as to the nature of the case Dr. Mesnet reports, I may say that I have notes of cases in which after epileptic fits which began by chewing, &c., movements, or by a crude sensation of smell, there have been post-epileptic actions of great elaborateness during so-called unconsciousness.

movements (mâchillement, &c.) *Lancet*, January 14, 1899, from Herpin's *De l'Epilepsie*, p. 275, 1852; there is in Herpin's report of that case no mention of a dreamy state. I now give a quotation from Herpin's other work, *Des Accès*, &c. I do so to impress on the younger medical neurologists the great importance of observing a symptom which at first thought may seem to be of little value.[1]

Herpin wrote, *Des Accès Incomplets d'Epilepsie*, pp. 150-1, "En étudiant les débuts des attaques, nous avons décrit les symptômes qui se lient a ce spasme [le spasme pharyngé] sous les noms de mâchonnements, de mastication, de gustation, de déglutition, nous ne répéterons pas cette description, mais nous devons insister sur le fait *que ces signes sont précieux pour le vertige comitial qu'ils distinguent du vertige ordinaire d'une syncope* légère, d'un faible accès hysterique, &c. (No italics in original.) Ces phénomènes sont, chez quelques épileptiques, tellement caractérisés et constants, que les patients, au lieu de donner aux accès incomplets dans lesquels il se montrent, les noms, en quelque sorte génériques, d'absences, d'étourdissements, de fausses attaques, &c., les appellent *mouvements de bouche, mâchonnements, mâchillements, rincements de bouche.*" (Italics in original.)[2]

I give also part of what Herpin wrote on *perturbations de l'intelligence* (commonly called intellectual auræ), which I call dreamy states. It is a pity that he did not mention the particular "hallucinations" some of the patients had; I imagine these "hallucinations" were what I call crude sensations.

"Pour dix patients le début consistait en des *perturbations de l'intelligence*, avec ou sans hallucinations. Des exemples peuvent seuls donner une idée juste de ces aberrations; une malade d'un esprit très-distingué nous disait: 'Il s'agit d'un trouble purement intellectuel; je n'ai ni éblouissements, ni vertiges, je puis lire encore les mots, mais je n'en

[1] I do not think, I may say here, that Herpin distinguishes between what we now call the epigastric sensation warning and warnings by gastric spasm.

[2] One of my patients who had such movements at the onset of slight epileptic attacks (no dreamy state, but a temporary very elaborate quasi-somnambulistic state after his seizures), in a letter to me, spoke of his fits as "champing fits."

comprends plus le sens. C'est un état des plus pénibles; il me semble qu'une partie de mon intelligence assiste à l'égarement de l'autre.' . . . Un jeune prêtre de grande espérance s'exprimait ainsi: 'Ce sont des idées sans suite et cependant contradictoires; si j'entends une conversation, elle me suscite deux idées qui se combattent; je perds la tête et non pas la connaissance; j'entends, je comprends, mais mon esprit est ailleurs.' Autre version d'un autre patient: 'Il y a en moi deux personnes dont l'une jouit de la raison et l'autre déraisonne.' Un épileptique parlait d'une idée fixée, toujours la même, mais qu'il n'a jamais pu se rappeler. Un autre mentionnait seulement un égarement de la raison. Une jeune personne d'un esprit très-observateur racontait ainsi le début de ses attaques: 'Je suis prise, sans cause, d'une tristesse subite, et a l'instant même mes yeux restent fixés sur un objet et ma pensée sur une idée qui me rappelle l'image très-nette d'un ancien rêve; l'idée fixe m'absorbe tellement que, quoique je regarde toujours vers le même point, je ne vois plus l'objet.'"—*Des Accès Incomplets d'Epilepsie*, 1867, p. 111.

One reason for giving these quotations is that some of the symptoms I have mentioned here and in other papers as occurring in some slight epileptic paroxysms, seem so odd that the reports of the cases may appear to some medical men more like collections of scientific curiosities than things with practical bearing. I wish to show by the quotations from Herpin that statements essentially the same as those I make were made by a great authority long ago. I will here quote from a former article, BRAIN, July, 1888, p. 187.[1]

"Before leaving this part of my subject, I remark, by way of recapitulation, that he who neglects the 'dreamy state,' because it is indefinite and 'merely curious,' and such symptoms as chewing, &c., movements, and apparent alteration in the size and distance of external objects, because they seem trifling things, may not even surmise that his patient has the serious disease, epilepsy, in a rudimentary form, until a severe fit comes to tells him so."

[1] "On a Particular Variety of Epilepsy ('Intellectual Aura'): One case with Symptoms of Organic Brain Disease."

Remarks by Dr. Purves Stewart.

In the above case the history of gradual mental impairment followed by attacks of unconsciousness, together with the presence of headache, of optic neuritis, and of slight hemiplegia, pointed to the existence of gross intra-cranial disease. The left-sided weakness, most marked in the face, but also distinct in the arm and leg, indicated clearly as the site of the lesion some part of the motor tract, or of its vicinity, in the right cerebral hemisphere. The absence of hemianæsthesia and of hemianopia showed that the lesion was probably not in the neighbourhood of the internal capsule, but that it was situated more towards the cortex of the brain, a view which strengthened by the presence of "cortical anæsthesia" of the left hand. The occurrence of Babinski's extensor plantar reflex—extension of the hallux instead of the normal flexion, on tickling the sole of the foot—was not sufficiently constant to be of much significance.

The peculiar nature of the paroxysms, and especially their association with a sensation of smell, pointed to some abnormal condition of the temporo-sphenoidal lobe.

Drs. Hughlings Jackson and Beevor[1] record the case of a sarcoma, the size of a tangerine orange, situated at the most anterior extremity of the right temporo-sphenoidal lobe. During life the patient had left hemiplegia without hemianopia, hemianæsthesia, or affection of smell, taste, or hearing. She also had numerous fits with an "intellectual aura" and a "horrid smell of dirty burning stuff."

Drs. Hughlings Jackson and Colman[2] also record a case of a small subcortical patch of softening in the left uncinate gyrus. The patient had been subject to epileptic attacks accompanied by a "dreamy" state, with smacking movements of tongue, lips, and jaw, but without crude sensations of taste or smell.

Dr. James Anderson[3] published a case of a large basal cystic sarcoma arising from the pituitary body and spreading into the left temporo-sphenoidal lobe. It had given rise to attacks of peculiar sensation in the right hand, preceded by a sensation of a bitter taste in the mouth, with occasionally

also a peculiar smell, and accompanied by the "dreamy state." There were never any chewing or smacking movements of the lips. Smell was impaired on the left side, and there was slight defect of taste on both sides, especially the left. From affection of the left optic nerve, optic chiasma, and optic tract, the patient had blindness of the left eye with temporal hemianopia in the right.

Mr. Nettleship[4] also records a case of tumour of the pituitary body implicating the left optic nerve, chiasma, and tract, as well as the left temporo-sphenoidal lobe. In that case there had been paroxysmal sensations of suffocation in the nose and mouth. There is no mention of a dreamy state in that case.

Sanders[5] records a case of a large glioma on the under surface of the brain, involving the anterior part of the left temporo-sphenoidal lobe and the lower convolutions of the left frontal lobe, and destroying the left olfactory tract. The patient had fits, preceded by a warning of a "dreadful disagreeable smell," also chewing movements of the jaw and spitting of saliva. Later he had convulsions affecting the face, but not the limbs. No dreamy state was recorded in that case.

Dr. McLane Hamilton[6] also published a case of localised chronic pachymeningitis affecting the right uncinate gyrus and part of the adjacent convolutions, but without involvement of the olfactory bulbs. The patient had been subject to attacks, preceded by a peculiar disagreeable odour, either of smoke or of a fœtid character. No mention was made of any dreamy state.

In our case of A. B. the view as to the localisation of the disease in the right temporo-sphenoidal lobe was further supported by the impairment of hearing in the left ear without any discoverable cause in the ear itself, and, therefore, possibly referable to some slight affection of Ferrier's auditory centre on the side of the lesion.

The slight "cortical anæsthesia," the slight hemiplegia, most marked in the face, together with the occurrence of localised fits in the left arm and hand before death, are facts which, so far as they go, indicate either that the lesion was

not confined to the temporo-sphenoidal lobe alone, and extended to the lower central convolutions so as to affect the face and arm centres, or, more probably, that it pressed upwards in that direction.

The absence of an autopsy unfortunately leaves undetermined the exact nature and limits of the lesion. On the whole, the slow course and progressive march of the symptoms indicate that it was a new growth of some sort. The history of septicæmia rendered it conceivable that a chronic abscess might have been present. Mr. Ballance informs us that he has known a brain abscess to lie latent for fifteen months after an attack of septicæmia, without previous otorrhœa or other apparent local exciting cause, at the end of which period symptoms appeared for the first time, and rapidly proved fatal. But in A. B.'s case the symptoms were those of a lesion involving not only the temporo-sphenoidal, but also the lower central region, and this with very slight motor weakness, less in amount than an abscess of such size would have been likely to produce. It is, therefore, more probable that there was an infiltrating growth involving the temporo-sphenoidal lobe and the adjacent part of the "central" region. This, however, is mere hypothesis, and the absence of an autopsy is much to be regretted.

Amongst other points of interest in the case is the unilateral optic neuritis present on the side of the cerebral lesion. Mr. Marcus Gunn,[7] in a discussion on the diagnosis of brain tumours, states that unilateral, or preponderatingly unilateral, neuritis is, on the whole, in favour of the tumour being on the same side as that on which the neuritis is more intense, if other reasons exist for localising the tumour far forward in the brain. Our patient's case, so far as it goes, seems to bear out Mr. Gunn's views. But the rule is not an invariable one, for I have seen a case of a large frontal tumour in which the optic neuritis was both earlier and more intense on the opposite side from the lesion.

The remarkably large appetite, which our patient exhibited up to the last, is of interest when considered with a case of temporo-sphenoidal abscess and a series of cases of head injuries published by Mr. Stephen Paget,[8] in which voracious hunger or thirst were outstanding symptoms.

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(7) BRAIN, vol. xxi., 1898, p. 337.
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REMARKS ON LOSS OF MOVEMENTS OF THE INTERCOSTAL MUSCLES IN SOME CASES OF SURGICAL ANÆSTHESIA BY CHLOROFORM AND ETHER.

BY J. HUGHLINGS JACKSON, M.D., F.R.S.

AND

JAMES S. COLLIER, M.D., B.SC., M.R.C.P.LOND.

PART I.[1]

IN the *Lancet*, January 2, 1897 (Neurological Fragments, No. 17), I mention, in a footnote, the condition of the respiratory apparatus in some cases of surgical anæsthesia. A few years ago, Dr. Howard Sturge, my then house physician at the London Hospital, told me he had observed that a woman's breathing became abdominal when she was under chloroform. (By abdominal breathing in this note is meant respiration in which the diaphragm acts without the co-operation of the intercostal muscles, there being no respiratory movements of those muscles.) I have, since hearing Dr. Sturge's remark, observed abdominal breathing during surgical anæsthesia in several cases, both in men and women; the abdominal breathing is more striking in women because their normal respiration is chiefly costal. I learn from Dr. Hewitt's very interesting work "Anæsthetics and their Administration," p. 219, that "Snow stated that thoracic movements usually cease before the abdominal." (Dr. Hewitt is speaking of an over-dose of chloroform.) Abdominal breathing occurs in some cases of coma (apoplexy) in men and women from other causes than chloroform and ether.

[1] This part is written by J. H. J.

In many cases of apoplexy the breathing is mainly costal, the epigastrium sinking in inspiration. Probably the manner of respiration differs much according to the stage of coma in apoplexy as it does according to the stage of surgical anæsthesia. In this note I speak only of abdominal breathing in the coma of surgical anæsthesia. I much regret that I have not noted in all the cases I have observed, whether the anæsthetic used was chloroform or ether, or some mixture of anæsthetics.

Whether the breathing becomes abdominal or not probably depends most on the degree of the anæsthesia. I will mention two cases in illustration of this remark. I saw with Mr. Ballance and Dr. Collier a girl, 19 years of age, who had chloroform for a short examination (functional affection of one hip); during the anæsthesia her intercostals acted in normal fashion during respiration. I suppose the anæsthesia in this case was not nearly so deep as that in the following: I saw a young man made deeply anæsthetic with chloroform by Dr. Dudley Buxton for an operation of trephining the skull by Mr. Horsley; in this patient the intercostals did not act; abdominal breathing was well marked and great, the diaphragm being in vigorous action. We have to consider stages of anæsthesia. I have seen the breathing almost solely costal in men anæsthetised; possibly this was in an early stage of their anæsthesia. Dr. Collier will remark on this part of our subject.

In cases of surgical anæsthesia where the intercostal *muscles* do not act, there is, at any rate, loss of their respiratory *movements*. This may seem rather an absurd remark. But there are, I submit, non-respiratory as well as respiratory movements of these muscles. I have (N. F., No. 15, *Lancet*, February 23, 1895) drawn attention to the difference between their respiratory movements on the one hand, and their "voluntary" (cerebral, non-respiratory) movements on the other hand. In another note (N. F., No. 12, *Lancet*, December 22, 1894) I published a case (of supposed pneumonia) in which there was loss of the respiratory movements of the intercostal muscles with possibility of perfect "voluntary" (cerebral, non-respiratory) movement

of those same muscles. In that case these muscles did not act at all in the patient's respiration proper; his breathing was abdominal, and yet acted quite well in the "voluntary" (cerebral, non-respiratory) movement of drawing in his breath when told, the so-called (mis-called) "forced *inspiration*." I suggested (N. F., No. 12) that in this case there was loss of function, produced by pneumo-toxin, of the lateral horns of the cord (some centres of the costal part of what I call the Lowest Level of the central nervous system), from which resulted loss of the *respiratory* movements of the intercostal muscles; a further suggestion was that there was integrity of the remainder of the (costal) anterior horns, so that upon cerebral instigation this remainder acted effectually on the intercostal muscles in the "voluntary" movement of drawing in the breath, expanding the chest on command—a non-respiratory movement. It is possible that in the abdominal breathing of deep surgical anæsthesia from chloroform or ether, the lateral horns are affected earlier than, and that they are affected more than, the remainder of the anterior horns. But it is not likely that we could show that in these cases the remainder of the anterior horns is intact or but little affected, because no "voluntary" (cerebral) movements, ordinarily so-called, can, in the circumstances, be instigated from the cerebral cortex. Yet a patient may call out loudly when under chloroform or ether. In normal circumstances such calling out would imply prior inspiratory movement of the upper chest; rather, I ought to say (since drawing in the breath prior to vocalisation is a "voluntary," at any rate, a cerebral movement), it would imply expansion of the chest; it does not follow, however, that in surgical anæsthesia the movement prior to the vocalisation of calling out loudly, supposing there to occur such a movement, is costal; it may be diaphragmatic. It may be well to observe whether a movement of the intercostals for *expanding the chest* can occur in a patient so deeply anæsthetic by chloroform or ether that the *respiratory* movements of these muscles are lost. It will, however, most likely be held that the *quasi-voluntary* movement mentioned could not occur in so great a depth of surgical anæsthesia as that in which the *respiratory*

movements of the intercostals are lost. There is something more to be said here. Dr. Collier allows me to borrow from his remarks (see Part II.) what he writes on the case of a child deeply anæsthetised and breathing only with the diaphragm, that is in a condition in which the intercostals did not act in respiration. Dr. Collier writes: "When the operation was commenced the patient uttered two or three groans, each being preceded by an inspiratory movement in which the elevation-expansion movement of the chest was very marked."

There is another possible cause of loss of the movements of the intercostal muscles in deep anæsthesia to be considered. The late Dr. Franklin Hooper, of Boston, stated in a paper read before the American Laryngological Association, in 1885, that (now I quote from a lecture by Sir Felix Semon, *Brit. Med. Journal*, January 1, 1898), "stimulation of the recurrent laryngeal nerve, uncut or cut, with an electric current, which throughout was kept equally strong, resulted in adduction of the corresponding vocal cord when the narcosis—he always employed ether—was slight; whilst, when the anæsthetic was pushed to the condition of very deep narcosis, stimulation with a current of the same intensity resulted in decided abduction of the corresponding cord. This very extraordinary fact has, I am afraid, never met with the attention it deserves, although Mr. Horsley and I ('On an apparently Peripheral and Differential Action of Ether upon the Laryngeal Muscles,' *Brit. Med. Journal*, 1888) have specially pointed out its importance, after having convinced ourselves, by repetition of Dr. Hooper's experiments, of the correctness of his statements. The fact that the result of electrical stimulation—currents of the same strength being used throughout—entirely differs according to the depth of the narcosis, not only when the entire recurrent, but also when, after its section, its peripheral end is stimulated, clearly points to the conclusion that the difference cannot be due to any differentiating influence of the anæsthetic agent upon the nerve centres, but that there must be a specific peripheral and differential action on its part, through the agency of the

circulation, upon either the nerve-fibres, the nerve-endings, or the muscular substance itself. In other words, there must be biochemical differences between the two antagonistic apparatus, which in all probability some day will explain the fact of the greater vulnerability of the abductors as compared with the adductors."

So it is possible that in deep surgical anæsthesia the inaction of the intercostal muscles may be partly or mainly or solely owing to action of the drug upon the muscular tissue of the intercostals or on motor nerve endings in these muscles. If the intercostals are fully paralysed by such local action of ether, they could not act in any way, neither respiratorily nor in any possible quasi-voluntary expansion.

In chronic cases of disease of the nervous system paralysis of the intercostal muscles may be overlooked. This paralysis, at least if it develops slowly, does not seem to trouble a patient who is obliged to keep his bed by paralysis of the limbs or by other equivalent incapacity. I am not sure that some important characters of paralysis of the intercostal muscles are widely enough known. We must bear in mind that (Duchenne) the diaphragm can by itself, that is, I mean can, when the intercostal muscles are paralysed, raise and evert the lowest ribs. These ribs, diaphragmatic ribs Duchenne calls them, always move as mentioned, so far as I have yet seen, in cases of paralysis of the intercostal muscles from disease or injury of the spinal cord (with perhaps one exception in an infant), as well as in cases of that paralysis in surgical anæsthesia; they acted vigorously in the case of Mr. Horsley's chloroformed patient (*vide supra*). Action of the lowest ribs in these cases does not show, as the inexperienced might suppose, that the lowest intercostal muscles have escaped paralysis, and that only the upper ones have suffered. Faradisation of the phrenic nerve causes, among other effects, eversion and raising of the lowest ribs.

I think paralysis of the upper intercostals may be partly masked in surgical anæsthesia by great over-action of the scalene muscles. In a man deeply etherised by Dr. Buxton the diaphragm acted vigorously; the lowest and middle

parts of the sternum sank in inspiration, but the sides of the uppermost parts of the chest did not; in this case however the scalene muscles were in very great activity (the diaphragm and scalene muscles can act together in concertina fashion).

It must be well borne in mind that the upper chest wall does not stand still in all cases of paralysis of the intercostal muscles; in young people it may move a great deal. But in these cases the upper chest during respiration moves in a wrong way; it sinks in inspiration—sinks when it should rise—sinks when the diaphragm acts; so to say, the upper chest is "sucked in" in inspiration. The movement of the upper chest after this one of "sucking in"—the movement of the upper chest during relaxation of the diaphragm—is only recoil, and is in the opposite direction of the normal recoil in the expiration of the healthy. There is no muscular action of the intercostal muscles in either the "sucking in movement" or in the "return" movement, that by recoil. In some cases of this kind when respiration is rapid paralysis of the intercostals may not be easy to determine; the upper chest moves up and down very frequently, and the timing of its motions with the equally frequent actions and relaxations of the diaphragm may be difficult.

As to the mode of action of the diaphragm in causing movements of the lowest ribs, either in health or in cases of paralysis of the intercostals, I say nothing in this paper. I quote the following from Dr. G. V. Poore's "Selections from the Works of Duchenne," p. 325 (the reference is to Duchenne's *L'Electrisation Localisée*, 3rd ed., pp. 901-921):—

"(*A*) *Diaphragm.* . . . (1) My experiments on men and animals show that the contraction of the diaphragm caused by electrifying the phrenic nerve, isolated or otherwise, from its nervous connections (communiquant ou non avec l'arbre nerveux) causes an elevation and outward movement of the diaphragmatic ribs as long as the abdominal walls are intact. The antero-posterior diameter of the base of the thorax is also slightly increased. In some cases the outward movement of the lower ribs is communicated to the upper.

"This observation has been confirmed by clinical experience.

"(2) If the belly be opened and the bowels pulled downwards the contraction of the diaphragm by itself causes an *inward* movement of the diaphragmatic ribs."

I have not observed the movements of respiration of patients deeply under chloroform during the operation of abdominal section for the removal of large cysts, &c., and do not know whether in these cases the lowest ribs evert and rise or turn in.

I suggested to Dr. James Collier a further research on the respiratory condition during anæsthesia by ether and chloroform. An account of his observations follows.

Part II. [1]

At Dr. Hughlings Jackson's suggestion I have examined the respiratory movements in sixty patients, before and during the administration of anæsthetics for surgical purposes. Twenty-five of these patients were children, fifteen were male adults, and twenty female adults. In a few cases a graphic record of the thoracic and of the abdominal movements throughout the period of the administration of the anæsthetic was taken by means of two recording stethographs, one placed round the thorax and one round the abdomen. The anæsthetics used were chloroform or ether, and in one case first ether and then chloroform was administered. I am indebted to the kindness of Mr. Stansfield Collier for the opportunity of examining children during his operations at the Hospital for Sick Children, Great Ormond Street. The results obtained are here summarised.

(1) *Chloroform anæsthesia.*—During the initial stages of the anæsthesia the respiratory movements were increased in range and in frequency, and were either of the same type as before the administration of the anæsthetic or were more preponderatingly thoracic, and this increase of range and frequency persisted until the struggling ceased. The respiratory movements then slowly became of less range, and the

[1] This part is written by J. S. C.

elevation-expansion movement of the chest gradually became less and less, while the abdominal movement was more marked than before the anæsthetic was commenced.

At the period when the corneal reflex was no longer obtainable and when the anæsthesia was complete, the condition of the respiratory movements was generally as follows:—The elevation-expansion movement of the chest was very slight, while the abdominal (diaphragmatic) movement was well marked. In the female patients this condition contrasted most strikingly with the normal preponderatingly thoracic type of their respiratory movements.

After deep anæsthesia had lasted some time—a period which varied greatly with the individual, but which was on an average about fifteen minutes—the elevation-expansion movement of the thorax disappeared, though the scalene muscles still appeared to be in action, to a slight extent, in inspiration. Respiration was, with this exception, entirely abdominal. The eversion of the lower ribs in inspiration by the diaphragm was marked. The movement of the upper two-thirds of the chest alternated with the abdominal movement, the chest sinking in inspiration and rising in expiration. With a continuance of deep anæsthesia all trace of action of the scalene muscles disappeared. The diminution or cessation of the elevation-expansion movement of the chest seemed dependent, not upon the mere depth of the anæsthesia, but upon the time which deep anæsthesia had lasted. In these cases, after the anæsthetic was discontinued, the first elevation-expansion movement of the chest was noticed in the sigh of returning consciousness, after which the respiratory movements soon returned to their normal state. In cases where facial respiratory movements (action of dilatatores narium in inspiration) were present, such movements were lost early in the anæsthesia, generally just before the loss of the reflexes. In cases where the anæsthesia was of brief duration, though sufficiently deep for minor surgical procedures, very little diminution of the expansion movement of the chest in inspiration was noticed. A similar result obtained in subjects addicted to alcohol, and in these patients, even after prolonged deep anæsthesia,

the elevation-expansion movement of the chest on inspiration was not lost, but only diminished. The susceptibility of different individuals to chloroform, as evidenced by the change in the character of the respiratory movements, seemed to vary greatly.

(2) *Ether anæsthesia.*—The results obtained were similar to those obtained with chloroform, with this important difference, that the elevation-expansion movement of the thorax in inspiration, though much diminished after prolonged ether anæsthesia, was never completely lost in any case that I have had the opportunity of examining. In one case, after deep anæsthesia had been kept up by ether for twenty minutes, the expansion of the chest in inspiration was still obvious. Chloroform was then substituted for the ether, with the effect that in about two minutes the expansion movement of the chest in inspiration became completely lost.

(3) *Chloroform anæsthesia in children.*—The observed cases seemed to fall into three groups, according to the length of administration of the anæsthetic and the susceptibility of the individual.

(a) The elevation-expansion movement of the chest was greatly lessened, but was still present throughout the anæsthesia. (Brief anæsthesia.)

(b) The diaphragmatic movement and the elevation of the upper chest by the scaleni was marked. Sinking in of the supra-clavicular and supra-sternal fossæ was marked. This seemed to be due to an increased action of the scaleni, the first rib being drawn up farther than in normal respiration, and so making these fossæ deeper. The expansion of the chest, except that produced upon the lower ribs by the diaphragm was lost. The middle region of the chest sank passively during inspiration, the scaleni and diaphragm acting upon the thorax in concertina fashion. (Deep anæsthesia.)

(c) The inspiratory movement was solely diaphragmatic, the only thoracic movement being a slight raising and an eversion of the lower ribs. The abdominal inspiratory movement was accompanied by passive sinking of the chest and the expiratory movement by recoil expansion of the

chest. (Deep and prolonged anæsthesia.) In two cases of this group, the anæsthesia was apparently deep and the corneal reflex was lost. The diaphragm was alone in action in the respiratory movement. When the operation was commenced the patient uttered two or three groans, each being preceded by an inspiratory movement in which the elevation expansion movement of the chest was very marked.

The action of the diaphragm upon the lower ribs.—By the kindness of Dr. Voelcker I have had the opportunity of examining the action of the diaphragm and the effect of its contraction upon the lower ribs in a case where the abdominal *point d'appui* of the diaphragm had been removed by an extensive abdominal incision and the removal of a very large ovarian cyst. Duchenne[1] has proved that contraction of the diaphragm raises and everts the lower ribs, and, relying upon experimental simulation of the phrenic nerves in animals, states that the diaphragm only elevates the lower ribs so long as it is supported by the abdominal viscera and parietes, and that when, in one of the lower animals, the abdomen is eviscerated, contraction of the diaphragm is accompanied by depression and drawing-in of the lower ribs; and he rejects the opinion of Beau and Massiat[2] that the pericardium, &c., can afford a more or less fixed point from which the diaphragm can raise the lower ribs in its contraction.

In Dr. Voelcker's patient, a girl of nine years old, a laparotomy was performed for abdominal tumour, and an ovarian cyst, the size of a large melon, was removed.

Before the anæsthetic (chloroform) was commenced, the respiratory movements were preponderatingly thoracic, and some diaphragmatic movement, with eversion of the lower ribs during inspiration, more marked on the left side, occurred. When anæsthesia was complete the scalenes and diaphragm alone acted, the "concertina action" of the thorax being marked.

[1] *Physiologie des Mouvements*, 1867, p. 632.

[2] "Recherches sur la respiration," *Arch. génér. de Méd.*, 4e série, t. ii., Art. 3e.

A long incision was made in the middle line of the abdomen and the tumour turned out of the abdominal cavity whole, and removed, its pedicle being ligatured. The intestines were collapsed, and the abdominal walls were quite flaccid.

In this condition the eversion of the lower ribs during inspiration was very marked, although the upper ribs sank during inspiration, and this continued so long as the patient was observed. According to Duchenne's explanation of the mode of action of the diaphragm in raising the lower ribs the reverse should have happened, as the anatomical conditions were similar to those in his experiments upon eviscerated horses.

The condition in this case suggests that there may be some justification for the opinion of Beau and Massiat.

Part III.

We have to thank Dr. Risien Russell for the opportunity of observing the following experiments upon animals performed by him at the suggestion of one of us (J. H. J.) for the elucidation of this subject:—

Experiment I. Female Macacus rhesus. Ether anæsthesia.—In the earliest stage of the anæsthesia the respiratory movements were quiet and chiefly abdominal. There was a distinct elevation-expansion movement of the whole chest during inspiration, and there was marked eversion of the lower ribs during inspiration. With deeper anæsthesia, but before the corneal reflex was lost, the abdominal respiratory movements became much exaggerated. (Dr. Russell informs us that this is the rule in the lower animals.) At this stage the chest, with the exception of the lowest ribs, sank during inspiration. The supra-clavicular regions and the upper intercostal spaces were "sucked in" during inspiration. This sinking of the chest was certainly due, as Dr. Russell suggested, to the aspiratory effect of the strongly acting diaphragm upon the comparatively soft ribs of the monkey, and this aspiratory effect entirely masked any elevation-expansion movement. The stage of anæsthesia at which this occurred was an early one, and at the corresponding stage of ether anæsthesia in the human subject no thoracic paralysis is met with. When complete anæsthesia was reached diaphragmatic movement became quiet

and regular, the chest sank quietly during inspiration; there was, indeed, no trace of any elevation-expansion during inspiration, but the lower ribs were markedly everted.

In order to exclude the possibility of any obstruction in the larynx producing the peculiar respiratory movements mentioned, the trachea was divided and a canula was introduced. The abnormal respiratory movements remained unaltered.

The anæsthesia was then allowed to pass off until the elevation-expansion movement of the chest during inspiration returned. More ether was once more administered; on the deeper anæsthesia produced the same sequence of events as occurred on the first etherisation was again observed; first of all, exaggerated diaphragmatic action with passive "sucking down" of the upper chest during inspiration; and next, quiet diaphragmatic action with eversion of the lower ribs and gentle sinking of the thorax during inspiration.

The ether anæsthesia was again allowed to pass off, when the elevation-expansion movement of the chest during inspiration returned. Chloroform was then administered, and the same effect was observed as with ether, but it was much more rapid. During deep anæsthesia the trachea was clamped, and a few inspiratory movements preceded death.

Experiment II. Dog. Ether anæsthesia.—During the earliest stage of the anæsthesia the elevation-expansion movement of the thorax in inspiration became exaggerated. During the next stage—that in which there is rigidity, this movement was little marked owing perhaps to the rigidity of the trunk muscles (abdominal, thoracic and spinal muscles). With deeper anæsthesia when the rigidity had disappeared the thoracic elevation-expansion movement in inspiration again became exaggerated. With still deeper anæsthesia the thoracic expansion during inspiration ceased, and was replaced by sinking of the chest during inspiration; the movements of the chest and the abdomen alternating. The diaphragm was acting well, and the lower five ribs were markedly everted by the diaphragm.

Tracheotomy was now performed. The anæsthesia was allowed to diminish and ether was later again administered. The same sequence of events occurred as upon the etherisation before tracheotomy.

When in the stage of deep anæsthesia, the intercostal movements were lost, and whilst the chest and the abdomen were moving alternately, the paralysis of the intercostals quickly gave

place to marked intercostal action if the anæsthetic were discontinued for a short time.

The intercostal muscles were now exposed on one side and their condition observed both in slight and in deep degrees of anæsthesia. Under the first condition they could be seen and felt to be in vigorous action and in the second condition not to be acting at all.

The abdomen was then opened by a medium longitudinal incision from sternum to pubes, and the intestines were drawn out so as to do away with abdominal pressure. In this condition the anæsthesia was increased until there was complete loss of the elevation-expansion movement of the chest during inspiration, the abdominal and thoracic movements alternating, and yet notwithstanding the fact that all support had been removed from the abdominal surface of the diaphragm, the eversion of the lower ribs was as marked as before the abdomen was opened.

The effects of the chloroform were then observed, and were similar to, but more rapid than those of ether.

A CONTRIBUTION TO THE STUDY OF THE PYRAMIDAL TRACT IN THE CENTRAL NERVOUS SYSTEM OF MAN.

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(THE PHŒBE A. HEARST FOUNDATION.)

IN the March, 1898, number of the *Journal of Nervous and Mental Disease*, Dr. H. A. Hare published a case of unusual œdema in hemiplegia. The brain from this patient was given to me by Dr. Hare and Dr. Harris, the pathologist who performed the necropsy. I was unfortunately unable to obtain the spinal cord, and the tract that I am about to describe could not be followed, therefore, below the lower part of the medulla oblongata.

I examined carefully the brain, and in a horizontal section made at the level of the superior part of the optic thalamus and caudate nucleus I found a recent hemorrhage in the external capsule and lenticular nucleus of the left side. This hemorrhage appeared to be about the size of a hickory nut, and the brain substance around it was softened, more especially on the inner side of the focus. The anterior part of the posterior limb of the internal capsule was evidently involved in the softening, but the optic radiations, except perhaps in their most superior part, appeared, macroscopically, to be intact. In a section made horizontally through the left hemisphere, one inch below and parallel to the first section, no evidences of any lesion were seen.

The case was a very suitable one for the Marchi method,

as the patient had lived sufficiently long after the development of the hemiplegia for secondary degeneration to be well advanced.

I noticed in sections stained with osmic acid (Marchi's method) from the lower part of the medulla oblongata, a group of degenerated fibres lateral to the anterior horn. In tracing the tract towards its origin I used the greatest care to avoid mistake. The segments to be placed in Marchi's fluid of course had to be very thin, and to avoid the loss of a segment at any part where the tract might make a sudden bend, and, therefore, render the following of it difficult or impossible, I cut with the microtome each segment entirely before I took another piece from the medulla oblongata. In this way I knew the position of the tract in the segments as they were successively studied, and was able to trace the tract to its separation from the degenerated pyramidal fibres; and at a point where it did make an abrupt bend I could follow it without any danger of error. It is not altogether easy to trace an unknown bundle throughout its course by the Marchi method, for the loss of a small segment may lead to confusion.

A brief description of my findings was published in a discussion of a paper by Dr. E. L. Mellus, read before the Philadelphia Neurological Society.[1]

The origin of the tract could not be determined. Its fibres were mingled with those of the degenerated pyramidal fibres arising in the left hemisphere, and as the lesion that caused the degeneration was on a level with the upper part of the optic thalamus, the tract in question must have arisen as high as the situation of this lesion. It may have arisen in the lenticular nucleus, or thalamus, or it may have come from the cortex. Very slightly below the level of the exit of the fifth nerve from the pons, a band of fibres could be seen distinctly separating from the outermost and lateral portion of the left pyramidal tract. This separation was at first gradual, and the two bundles remained close together for a short distance. Lower in the pons the more dorsolateral of these two tracts passed quite abruptly backward

[1] SPILLER. *Journal of Nervous and Mental Disease*, March, 1899, p. 178.

and slightly laterally, and entered the trapezoid body. At this portion of its course, in a transverse section, the bundle in passing backward formed a long and narrow band of horizontal lines of black dots, showing that the fibres were cut longitudinally. At the junction of the medulla oblongata and the pons it took a position lateral to the uppermost portion of the inferior olive. As the inferior olive increased in size, the bundle passed backward, and where the olive had its greatest width the bundle was on the posterior and lateral side of the lower olive, at the periphery of the medulla oblongata. The bundle remained on the left side below the motor decussation, but it could not be followed below the level of the first cervical segment, as the spinal cord could not be examined. The degenerated fibres forming this tract were numerous. In the medulla oblongata the position of the tract was very similar to that of Gowers' bundle.

Gowers' tract has been studied by many investigators, but Hoche says that his two cases (1896) were the first in which the tract in adult man could be traced to its termination in the cerebellum. Patrick traced it as far as the corpora quadrigemina.

According to Hoche, the extent of Gowers' tract, as shown by secondary degeneration, depends very much on the location of the lesion producing this degeneration. When the lesion is situated low in the cord the degeneration does not extend above the cervical region. The method of staining also makes a very great difference, as the fibres in the upper part of the tract are so scattered in places that the loss of these fibres is not detected by the Weigert hematoxylin stain.

Hoche refers to the fact that Gowers could not trace the tract above the upper part of the cervical swelling, and the lesion in his (Gowers') case was in the upper portion of the lumbar swelling; and that Schaffer could not trace it by the Marchi method above the second cervical root. If further investigations confirm these observations, Hoche thinks it is probable that only the fibres of Gowers' tract from the upper and middle thirds of the spinal cord are in relation with the cerebellum, and that those from the lower third of

the cord connect cells within the cord at lower and higher levels, *i.e.*, those within the lumbar and sacral region with those within the cervical. If this view is correct it seems reasonable to suppose that descending fibres may be contained within Gowers' tract.

The course of Gowers' tract as described by Hoche is very different from that of the tract seen by me. In the first place, Hoche was describing ascending degeneration, from a lesion in the seventh thoracic segment in his first case, and I have observed descending degeneration. At the motor decussation the position of Gowers' tract as given by Hoche (fig. 3, case 1) corresponds with the position of my bundle. The position of the tract in figs. 5 and 6 is also similar to that of my bundle, but in fig. 7 (the level of the knee of the facial nerve) the resemblance ceases. From here on Gowers' tract, as described by Hoche, passes dorsally, and my tract passes ventrally.

Hoche could not trace Gowers' tract beyond the posterior corpora quadrigemina in either of his two cases, and he found that the tract turned upon itself and entered the cerebellum in such a way that in sections taken from the upper part of its course the tract was obtained at two different parts in the same section. The fibres of Gowers' tract entered the cerebellum partly in the velum medullare anticum, partly in the anterior cerebellar peduncle.

Below the primary lesion of the spinal cord in Hoche's first case, degenerated fibres were found in Gowers' column as far as within the lumbar region, but this does not prove that Gowers' tract degenerates downward.

The results obtained by Rossolimo in man are very different from those of Hoche.[1] Rossolimo[2] believed that Gowers' tract terminates in the posterior corpora quadrigemina, the substantia nigra and the globus pallidus. Quensel's[3] paper is in support of Hoche's findings in great part.

Figures 1, 2, 3, 4 and 5 in Bruce's[4] article show that the ventral cerebellar tract occupies a position similar to that of

[1] HOCHE. *Archiv für Psychiatrie*, vol. xxviii., 1896, p. 510.
[2] ROSSOLIMO. *Neurologisches Centralblatt*, 1898, p. 935.
[3] QUENSEL. *Neurologisches Centralblatt*, 1898, p. 482.
[4] BRUCE. BRAIN, vol. xxi., p. 374

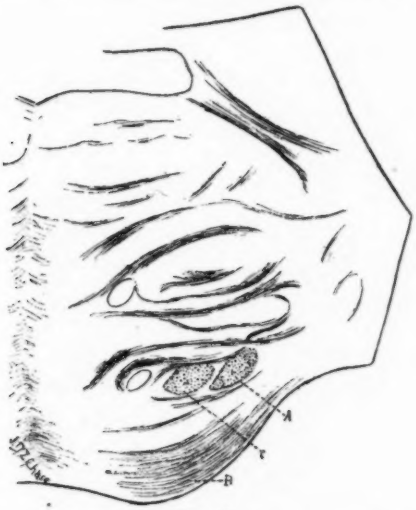

FIG. 6.

The degenerated bundle A has become separated from the adjoining fibres of the pyramidal tract in C. B, fibres of middle cerebellar peduncle. Fig. 6 represents the position of the bundle seen in fig. 1.

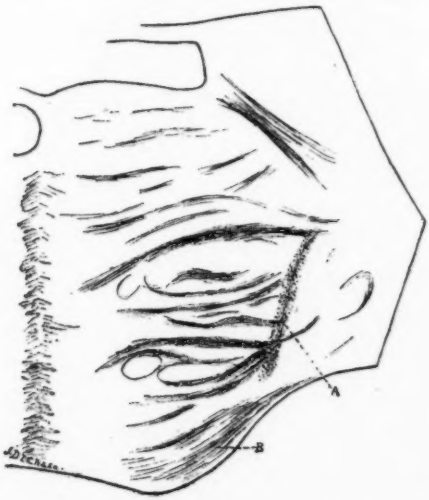

FIG. 7.

The bundle A is seen in longitudinal section. Fig. 7 represents the position of the bundle seen in fig. 2.

my bundle in the medulla oblongata, but in the pons the two tracts separate very widely.

In speaking of the afferent ventro-lateral tract, Russell[1] says that the position of this tract in the fifth and all other

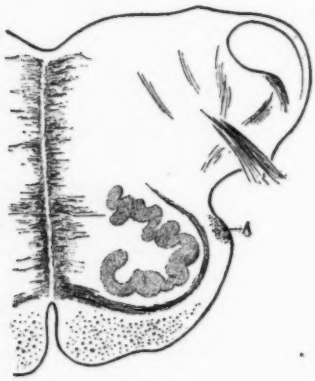

FIG. 8.

Fig. 8 shows the relation of the bundle A to the lower olive.

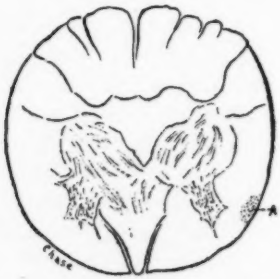

FIG. 9.

Fig. 9 shows the bundle A at the lower part of the motor decussation. Fig. 9 represents the position of the bundle seen in fig. 4.

segments of the cervical cord on the cephalic side of the lesion was more or less midway between the periphery of the cord and gray matter of the ventral horn, the band of fibres between this sclerosed area and the periphery of the cord

[1] RUSSELL. BRAIN, vol. xxi., p. 170.

being absolutely intact. This statement differs from those of most authors. My band of fibres in the upper cervical region is exactly on the periphery of the cord and this would seem to be a further proof that it is not a part of Gowers' tract.

Campbell,[1] in his excellent digest on the tracts of the spinal cord, says Gowers' tract degenerates in an ascending direction, and he makes no reference to a descending degeneration of this tract. V. Bechterew thinks it is probable that in some cases in which a descending degeneration of Gowers' tract has been observed, the degeneration was really in Loewenthal's anterior marginal bundle, although v. Bechterew[2] does not entirely exclude the possibility of descending fibres in the fasciculus antero-lateralis.

There seems to be considerable doubt as to whether Gowers' bundle does degenerate downward, and as far as I know no case has yet been reported in which this tract was degenerated in man as the result of a cerebral lesion.

I find an interesting statement authorised by Schäfer[3] in "Quain's Anatomy." It is said that in the monkey a few fibres of the antero-lateral column degenerate after lesions of the cerebral hemisphere of the opposite side. "They are mingled with those of the descending cerebellar tract and with those of the tract of Gowers, and are connected with cells in the Rolandic region of the cerebral cortex, as shown by the fact that they degenerate after lesions of that region. These fibres may perhaps be regarded as belonging to the system of the direct pyramidal tract, which in the monkey does not exist as a well-marked tract as in man: it has not yet been ascertained whether they occur in man as well as the direct pyramidal." These fibres are said to degenerate after a lesion of the *opposite* cerebral hemisphere.

Loewenthal[4] described two bundles in the lateral column

[1] CAMPBELL. BRAIN, vol. xx., p. 522.
[2] v. BECHTEREW. *Die Leitungsbahnen im Gehirn und Rückenmark*, second edition, pp. 82, 90.
[3] "Quain's Elements of Anatomy," vol. iii., part I., p. 25.
[4] LOEWENTHAL. *Revue Médicale de la Suisse Romande*, 1886, p. 529.

of the cord in the cat and dog; one situated in the dorsal segment of the lateral column he called the intermediate system of the lateral column (système intermédiaire du cordon latéral); the other, situated in the ventral segment of the antero-lateral column he called the anterior marginal bundle (faisceau marginal antérieur). He did not know the origin or termination of these tracts. This anterior marginal bundle might be thought to be the same as my tract, but recent investigations seem to indicate that Loewenthal's bundle comes from the cerebellum.

My tract could hardly be confounded with the intermediary system of Loewenthal. The major part of it does not occupy the mesial portion of the antero-lateral column in front of the lateral pyramidal tract. The literature on this tract is given by Campbell (*l.c.*) and v. Bechterew (*l.c.*).

The fibres in the peripheral part of the ground bundle of the antero-lateral column that degenerate downward, according to v. Bechterew, are the spinal portion of the posterior longitudinal bundle and the fibres from Meynert's decussation that arise in the anterior corpora quadrigemina; the fibres of Forel's decussation that arise in the lateral portion of the thalamus and the nucleus ruber; the vorderes Randbündel that arises in the cerebellum; and finally the olivary tract, coming from the lower olive (*l.c.*, p. 80).

By vorderes Randbündel, v. Bechterew means Loewenthal's faisceau marginal antérieur. From the investigations of Marchi, Biedl, and Basilewski (the latter in v. Bechterew's laboratory) v. Bechterew concludes that this tract arises in the cerebellum.

The tract described by v. Bechterew,[1] Held,[2] and Boyce,[3] as arising in the anterior corpora quadrigemina, certainly could not be confused with my tract, and its location within the spinal cord is entirely different from that of the fibres observed by me. The tract arising in the thalamus is also very different (Boyce, v. Bechterew).

Risien Russell, in experimenting on dogs and monkeys,

[1] v. Bechterew. *Neurologisches Centralblatt*, 1897, p. 1074.
[2] Held. *Archiv f. Anatomie u. Physiologie. Anatom. Abth.*, 1893, p. 239
[3] Boyce. *Neurologisches Centralblatt*, 1894, p. 466.

cut the lateral portion of the medulla oblongata between the "ascending" root of the fifth nerve and the inferior olive, and observed descending degeneration in the antero-lateral column. He regarded this tract as the same as that described by Boyce. This tract observed by Risien Russell differs somewhat from the one seen by me. I can only compare the cervical region in each case, as I was unable to trace my tract below this region and Russell did not trace his above, on account of the location of the primary lesion. The fibres in my case occupied a very small portion of the area represented as degenerated in Russell's figure 2, and if Russell's tract was identical with that described by Boyce, as Russell believed, then its origin was very different from the origin of my tract. None of the degenerated fibres in my case were situated just ventrally to the crossed pyramidal tract and internally to the anterior part of the direct ascending cerebellar tract, as Russell[1] described in his case.

My tract cannot be confused with that described by Marchi, Biedl,[2] and Thomas,[3] as arising in the cerebellum; although its position may be similar, its origin distinguishes it at once. It cannot be confused with the tract resulting from injury of Deiters' nucleus and other structures in the lateral region of the medulla oblongata (Ferrier and Turner,[4] Risien Russell[5]) for the same reason; or with the fibres seen by Mott.[6]

Risien Russell has described a tract situated not very far from the position of my bundle. The case was one of tumour of the cerebrum. In addition to the degenerated pyramidal fibres studied by the Marchi method, he found another and quite distinct tract of degeneration seen on the side of the lesion, and occupying the ventral margin of the medulla, just outside and opposite to the ventro-external angle of the inferior olive. It was a well-defined band of

[1] Russell. Brain, vol. xx., p. 409.
[2] Biedl. *Neurologisches Cetralblatt*, 1895, p. 434.
[3] Thomas. *Le Cervelet.*
[4] Ferrier and Turner. *Phil. Trans. Royal Soc.*, 1894, vol. clxxxv., B.
[5] Russell. Brain, vol. xx.
[6] Mott. Brain, 1895.

degeneration, quite distinct and separate from the degenerated pyramid, lying between the external arcuate fibres as they course round the medulla opposite the inferior olive. Russell[1] was unable to establish a connection of this tract with any of the degenerated fibres met with in the pons. A little lower in the medulla oblongata this tract was found in the lateral region opposite the ventral tip of the anterior horn. He believed that this tract was not derived from the degenerated pyramid.

This tract observed by Russell does not seem to be the same as the one observed by me. Russell was unable to satisfy himself as to the precise point of origin of his fibres. The position of the tract was not that occupied by the degenerated fibres in my case. It was at the periphery of the medulla oblongata and opposite the ventro-external angle of the inferior olive. My tract was also at the periphery but dorso-external to the inferior olive, and at some distance from the place occupied by Russell's fibres. In the cervical region Russell's tract seems to have occupied a more anterior position than mine.

In Thomas'[2] recent review of certain descending tracts in the antero-lateral column I do not find the description of any tract exactly similar to mine.

I believe that the tract described in this paper has hitherto been unrecorded. If I am in error in this I shall cheerfully accept correction. One case may not be sufficient to establish the existence of an unknown tract, and should confirmation of my findings not be granted, I may have to assume that the bundle of fibres seen by me was an aberrant bundle from the pyramidal tract, and not usually present in the central nervous system. This explanation does not seem probable, but I mention it in order to be as guarded as possible in all statements relating to scientific matters.

There are a few more tracts described by others that might be mentioned in this connection, but the resemblance to my bundle is not very great; such, for example, as the tract coming from the lateral fillet observed by Ferrier

[1] Russell. Brain, vol. xxi., p. 145.
[2] Thomas. *Journal de Physiologie et de Pathologie Générale*, No. 1, 1899.

and Turner,[1] or v. Monakow's[2] "aberrirendes Seitenstrangbündel" or Pick's bundle, which has been found by Hoche[3] to degenerate downward.

The existence of homolateral fibres in the crossed pyramidal tract on the side of the cerebral lesion has been noted by a number of observers. The origin of these fibres in the degenerated pyramid of the same side seems to have been first observed in 1894 in a cat by Russell.[4] The limited literature on the subject is given by him. I may add a reference to the paper by Dejerine and Long,[5] and one to the paper by Redlich.[6] In my preparations the origin of these homolateral fibres in the degenerated pyramid was distinct.

Dejerine and Long (*l.c.*) in cases of hemiplegia, studied by the Marchi method, observed degenerated fibres that left the anterior pyramid, took a horizontal course, mingled with the external anterior arciform fibres, passed in front of the arcuate nucleus, and decussated in the median raphe. These fibres were not traced beyond the interolivary layer of the opposite side. Similar fibres are also described by Redlich (*l.c.*) in experimental work.

The brain from a case of cerebral tumour was given to me by Dr. C. W. Burr. It was studied by the Marchi method. In addition to the usual degeneration of the pyramidal tract, I found degeneration of the postero-external columns, of equal intensity on the two sides, and the degeneration of the external anterior arciform fibres, described by Dejerine and Long, was very distinct. I was unable to follow these fibres beyond the interolivary layer of the opposite side.

I am indebted to Drs. C. Y. White and S. S. Kneass, associates in the Pepper Laboratory, for the photographs, and to Mrs. P. P. Chase for the drawings.

[1] Ferrier and Turner. *Phil. Trans. Roy. Soc.*, 1894, vol. clxxxv. B.
[2] v. Monakow. *Archiv f. Psychiatrie*, vols. xiv. and xxii.
[3] Hoche. *Archiv für Psychiatrie*, vol. xxx., 1898.
[4] Russell. Brain, vol. xxi., p. 145.
[5] Dejerine and Long. *Comptes rendus de la Soc. de Biologie*, 1898, p. 864.
[6] Redlich. *Neurologisches Centralblatt*, 1897.

DESCRIPTION OF FIGURES.

FIG. 1.

The bundle A is separated as a distinct band of degenerated fibres, and is seen in transverse section; at a little higher level it is united with the portion of the pyramidal tract designated by D. Fibres of the middle cerebellar peduncle are seen on the extreme right of the photograph.

FIG. 2.

The bundle A is seen in longitudinal section, with a low-power lens. It begins at this level to pass posteriorly. By mistake it is represented on the right side of the pons instead of on the left. Fibres of the middle cerebellar peduncle are seen at the lower left corner of the photograph.

FIG. 3.

(Same as fig. 2.) The bundle A is seen under higher magnification, and the degree of degeneration, as indicated by Marchi's method, is shown.

FIG. 4.

The bundle A is shown under low-power at the periphery of the cord, extending about as far forwards as the anterior end of the anterior horn (E). The anterior horn is seen on the left of the photograph. The bundle A is, by mistake, represented on the right side of the cord instead of on the left.

FIG. 5.

(Same as fig. 4.) The bundle A, which is somewhat indistinct in fig. 4, on account of the low-power lens employed, is seen in this photograph under higher magnification.

Figures 6, 7, 8, and 9 are diagrams showing the relative position of the bundle A.

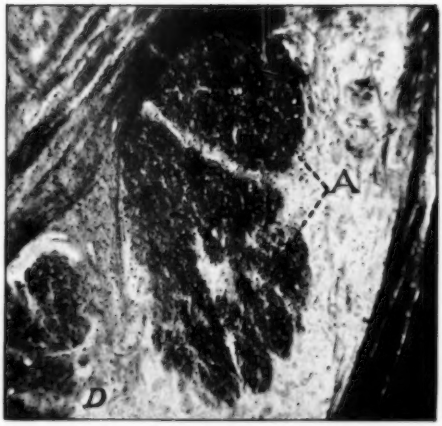

FIG. 1.

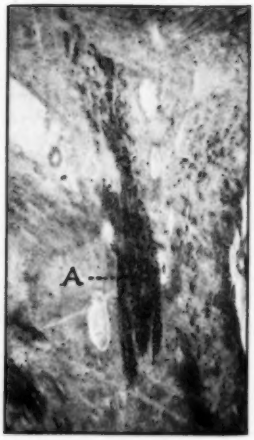

FIG. 2.

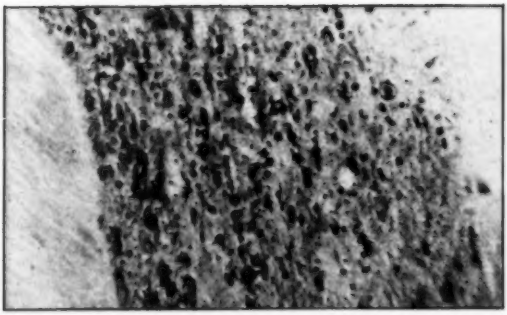

FIG. 3.

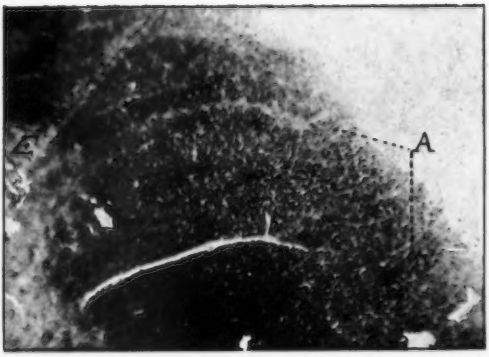

FIG. 4.

FIG. 5.

NOTE ON A FORM OF DEMENTIA ASSOCIATED WITH A DEFINITE CHANGE IN THE APPEARANCE OF THE PYRAMIDAL AND GIANT-CELLS OF THE BRAIN.

BY JOHN TURNER, M.B.ABER.

I WISH to draw attention to certain cases of mental disease which present a definite pathological anatomy and of which I have not been able to find any previous description.

The lesion shows itself chiefly in the nerve-cells and appears to be of widespread distribution, attacking the great majority of the pyramidal nerve-cells in all parts of the cerebral cortex (frontal, ascending-frontal, occipital being the regions examined).

It is especially well marked in the giant nerve-cells of the ascending convolutions, and for this reason I have chosen cells from this part for my description and photographs.

It is not a common change in insanity, as in considerably over a hundred brains of lunatics examined by Nissl's method or modifications I have besides the cases here noted only seen one or two individual cells so affected.

In the case of a man whose leg had been long amputated, no cells of this description were found in the opposite leg centre of the cortex.

The lesion appears to be identical with that described by Nissl and others after section of the axon of a motor nerve-cell, and by Barker in the forehorn-cells in cases of epidemic cerebro-spinal meningitis (see *British Medical Journal*, December 26, 1897); but it is questionable how far this similarity extends; at any rate, with osmic acid, very

definite alterations of structure are shown both in nerve-cell and its axis-cylinder continuation, which point to a probably degenerating state of the neuron. Although in one of the seven cases there was no mention made of a condition of dementia, but only prolonged and profound melancholia, I am inclined to think that the lesion is the anatomical groundwork of the dementia, which is often rapid in its course and probably always fatal.

Cases Nos. 1 and 2 are of interest, as with a clinical history resembling to a certain extent general paralysis in its acuter forms, the autopsy revealed naked-eye appearances quite distinct from general paralysis, neither did the microscopical investigation show any features in common with this latter disease.

As regards the melancholia case (No. 5), it is to be noted that she contracted a malignant form of diphtheria affecting the whole of her œsophagus and to which she succumbed after an illness of a fortnight.

In case No. 4 I was able from the symptoms to foresee the nature of the changes in the nerve-cells.

Certain symptoms were present in most of the cases, and of these the most obvious was paresis. The patient, for no obvious reason, occasionally fell down whilst walking, and ultimately there was complete inability to walk. Possibly the weakness of the arms was as great, but was not so obviously developed.

When the notes of the cases do not mention incapability of walking, there is mention of involuntary jerkings of the limbs. The demented condition of the patient prevented any greater precision in testing their movements, and the same cause interfered largely in endeavouring to determine sensory defects. The sensibility to pricking (painful impressions), in the cases where any reliance could be placed on the results, was greatly blunted, or entirely absent, but generally more marked in the lower extremities.

The pupils reacted to light and for accommodation to the end. The knee-jerks were exaggerated; plantar reflexes absent; tongue free from tremor; articulation not generally interfered with.

The arterial tension of three cases was high (= 150 to 210 mm. Hg.). This is interesting in view of the fact that a thickening of the arterioles of the cortex and minute cerebral hæmorrhages were found in all the cases.

Diarrhœa was very commonly noted towards the end.

The whole case may run a fairly rapid course, varying from a few to eighteen months, or, after prolonged mental disorder (melancholia so far only noted), dementia sets in and death as a rule rapidly ensues.

Pathological Anatomy.

A reference to the cases will show the leading naked-eye changes noted in the head and viscera. The brain may present a fairly healthy appearance, or there may be merely evidence of atrophy in the frontal convolutions. In others the meninges are opaque and thick over the vertex, but not adherent, and, microscopically, this thickening is seen to be an increase in the connective tissue of the arachnoid, and quite unattended with any round-cell infiltration of the pia.

A general feature noted microscopically in all the sections examined was a thickening of the arterioles and capillaries of the cortex. Sometimes the lumen was nearly obliterated. In the arterioles this thickening appears to be due to increase of the muscularis and adventitia; in the capillaries, to a fibroid thickening. Probably, in both cases, this latter predominates, for when stained in picro-fuchsin, the thickened vessels stand out as prominent red rings or lines. Tortuosity was also noted to a very considerable extent. Minute hæmorrhages into the cortex (microscopical) were seen in all the cases, sometimes five or six in one section.

The larger vessels of the white matter often had a copious collection of colloid bodies lying in their immediate neighbourhood. They showed a blue centre fading into a red circumference when stained with acid fuchsin and toluidin blue.

There was never any appearance of spider-cells or proliferation of neuroglial fibrils.

Nerve cells.—As noted before, the change in these is

widespread, and appears to affect the majority of all the pyramidal cells, and is especially well seen in the giant-cells.

Fresh squeezed-out portions of cortex, coloured in methylin blue or toluidin blue, show the change with the greatest clearness, and my experience, contrary to that of Drs. Robertson and Orr, is that most pathological changes are better seen by this method, when properly carried out, than in sections.

The cell is probably smaller than in its normal state, except when distended with pigment, and often somewhat globular; it has generally an irregular border staining deeply, and which often fails to show definite chromophile elements. The whole centre of the cell consists of a pale, homogeneous mass, having a blurred or washed-out appearance.

The nucleus, which is small and shrunken, stains a pale blue, and is situated at the periphery of the cell, or it may be up in its apex, or in the junction of a dendrite with the cell. It contains a dark, round or oval, and probably shrunken, nucleolus.

Generally a large part of the cell is occupied by a highly refractive bright yellow pigment, and this may distend the cell enormously in one direction. This substance stains either a dark brown or jet black with osmic acid,[1] but that it is not wholly of a fatty nature is rendered probable by the fact that it is still seen in the cells in sections which have been subjected to chloroform and xylol. Generally a fair number of dendrites are given off from the cell, but they are, as a rule, unduly thin, and stain of a pale uniform blue, or else show a few attenuated chromophile threads.

The apex, at its junction with the cell-body, generally shows a few fine chromophile threads, but further along it does not stain at all, and is barely visible. Very often there is an abrupt line of demarcation separating the pale apex from the densely-stained cell-body border (see fig. 1).

In sections, fixed in sublimate and stained in toluidin blue, the appearance of the cell-body is much the same as in

[1] This refers to cells which have been hardened in osmic acid; in the case of films the pigment merely becomes slightly darker and takes on a dull greenish tint.

films; the pigment appears of a greenish colour, owing, probably, to the blending of its yellow with the blue stain. The nucleus is almost always shrunken, and, with a distorted outline, contains only a few loose granules and a dark nucleolus, and is almost always eccentrically placed—often, indeed, bulging from the cell-body. The pale, almost colourless, hyaline-looking body of the cell, which generally has a somewhat circular contour, gives these cells a most characteristic appearance; the pigment is often almost imperceptibly blended with it at its outer part. Even with a one-twelfth oil immersion, it appears homogeneous. In other respects, the description given of the whole cells applies to those seen in optical section.

Case No. 6, which was examined after fixing in osmic acid,[1] gave very interesting results.

The tangential fibres of the first layer were present in fairly large numbers, and showed a normal appearance in the motor region, but in the frontal regions they were practically absent.

In the motor region the great majority of the medullated nerve-fibres in the lower strata of the cortex and the adjacent white matter showed very marked changes; the staining, in general, was not so dark, owing to the fact of disappearance of some of the myelin, and that which remained was in the form of little separate black drops on either side of the nerve-fibre, which could be seen in its place more darkly stained than usual; and often in advanced cases of degeneration, when no myeline remained, it was nearly black. Fig. 4 shows a degenerated medullated fibre.

In certain parts of the section medullated nerve-fibres were seen, which gave a normal reaction with osmic acid,

[1] I find that a mixture of carbolic acid, 5 per cent., osmic acid 2 per cent., in equal parts, gives excellent results; it penetrates with great rapidity, fixes the smallest particles of degenerated myelin or fat, and also preserves the nerve-cells well. The pieces are left in it for twelve to twenty-four hours, then placed in 10 per cent. formaline for two days, passed into alcohol for one or two days, and then through chloroform and into paraffin. The section is fixed to the slide by capillarity, and then decolourised in permanganate of potash, followed by sulphurous acid. These sections counterstain very well with Ehrlich's acid hæmatoxylin, which is only allowed to act for a second on the section, and then washed off.

and some of these are shown in fig. 4, to contrast with the former figure.

Synopsis of Cases.

No. 1.—I. B., a female, admitted June 29, 1898, aged 45, married. Her father was a drunkard and died about 50 of apoplexy. Her mother is stated to have had several "fits," in one of which she died. Mother's sister melancholic. This is her first attack, of eighteen months' duration, and stated to be due to menopause and alcohol; it began with depression and occasional violent fits. She has been addicted to alcohol for years.

The facts certifying to her insanity were incoherent shouting and raving. On admission she was in feeble condition and weighed only 88 lbs. Temperature normal; complexion turgid; knee-jerks exaggerated; pulse (sitting), 158. Has conjunctivitis. Was restless and noisy; obstinate, paying no heed to questions. Dirty in habits; bad appetite. Parotitis of right side developed five days after admission. Teeth covered with sordes and mouth full of muco-purulent slime. Was in a delirious state, muttering, but occasionally replying relevantly. Her speech thick and indistinct. Plantar reflexes abolished. Was very restless, occasionally screaming, and had frequent attacks of involuntary jerking of the limbs. Became worse; comatose. Left pupil the larger. they both react slightly to light. Face covered with a greasy sweat; mouth open; eyes closed. On July 7 she died. Autopsy seventeen hours later. Body fairly nourished. Blood vessels at base normal. Meninges fairly clear and stript readily; they were heavily waterlogged over the anterior two-thirds. Lateral ventricles dilated and filled with blood-tinted serum. Atrophy of convolutions of anterior two-thirds. Consistency of brain was normal. No granulations in any of the ventricles. Pons and cerebellum appeared normal. Small patch of consolidation in left lung; old adhesions of right side. The cardiac muscle was rather soft; valves normal. Liver congested; spleen firm; kidneys slightly granular.

Weights in grammes.—Cerebrum (stript), 988; meninges, 20; cerebellum, 133; stem, 27; total, 1,168. Heart, 208; liver, 1,208; spleen, 128; right kidney, 143; left kidney, 165.

The idea was present that this was an acute case of general paralysis, but the entire absence of any of the pathological appearances, macroscopical and microscopical, showed conclusively that it was not.

No. 2.—J. W., a female, admitted October 15, 1897, widow.

This is her first attack, and is said to be of only ten weeks duration, following influenza. Has been a hard-working, sober woman. Aged 52.

The facts certifying to her insanity were: irrational conversation; at times morose, and says she is a lost woman; at others hilarious, laughing and singing immoderately. On admission she is thin, and weighs 93 lbs. Temperature normal. Dark sallow complexion, coarse skin. Pupils equal, and react normally to light and on accommodation. Knee-jerks exaggerated. Tongue free from tremor, furred. Slight cough. Nocturnal urine, acid, 1016; no albumen. Is excited, spits on the floor, and answers rudely when remonstrated with; sings when her chest is being examined; replies in a flippant, careless way, "don't know," to most questions. In December it is noted that, as a rule, she is excited one day and quiet the next. The left pupil is slightly the larger; they both react well to light and on accommodation.

March 24, 1898.—The day before yesterday she fell forwards on her face whilst walking round the farm; was not convulsed, and did not lose consciousness. This has happened several times recently. Is now in bed, talking to herself. Tongue clean, pulse 75, arterial tension = 150 mm. Hg.

April 2.—Up yesterday, but had to be put to bed again, as she could not stand. Clean in habits. Pulse 78, temperature normal, urine free from albumen, 1010.

April 20.—Up, walks unsteadily, and leans over to the right. Is now very lost and silly.

April 30.—Was yesterday very excited, rushing about the ward and falling about. To-day she keeps chattering a lot of nonsense in a chanting tone. Diarrhœa set in on May 16, and on the 26th she died.

Autopsy (twenty-five and a half hours later).—Poorly nourished. Basal vessels healthy. Meninges not opaque, stript readily, rather thick. No marked wasting of the convolutions; brain of normal consistency. Lateral ventricles dilated. No granulations on them or on fourth ventricle. Both lungs bound down by old adhesions. Heart normal, no atheroma. Liver pale and soft. Spleen firm. Kidneys small, soft, pale; cortex not thin; capsules stript readily.

Weights in grammes.—Cerebrum (stript), 1031; meninges, 20; cerebellum, 120; stem, 27; total, 1,198. Heart, 204; liver, 1,040; spleen, 132; right kidney, 92; left kidney, 76.

No. 3.—E. E. W., female, admitted November 6, 1895, baker's wife. First attack of eighteen years' duration. Her

father died of Bright's disease. She has a daughter insane. On admission she weighs 112 lbs., and is in fair health and condition. Knee-jerks exaggerated. Right pupil the larger; they react normally to light and on accommodation. Tongue slightly tremulous, protruded markedly to the right. Urine: specific gravity 1021, a faint trace of albumen. Her mental condition was one of delusional insanity; she imagined that some women were plotting against her and her husband, and conspiring to blow her up with dynamite. She was neat, quiet, and industrious, and gave no trouble, and remained thus till April, 1898, when she made an attempt on her life, and became acutely melancholic, restless, agitated, refusing food, and not able to sleep. Her bodily condition deteriorated greatly, she picked her face, looked miserably ill, and had to be held in bed to prevent her from jumping out and roaming in the ward.

May 17, 1898.—Pulse 102, regular; arterial pressure = 150 Mg. Hg. Knee-jerks exaggerated. Tongue clean. Temperature normal. Is very agitated and looks haggard.

During July she suffered off and on from diarrhœa, and had dermatitis of face and back of hands (probably from the sun). Mentally more composed.

In September she rapidly became demented, dirty in her habits, and very greedy. By November she was bed-ridden, and had lost the use of her legs, was filthy in her habits, very restless, and often falling about.

December 8.—Lies on her right side, and exhibits involuntary jerking movements of arms and legs, and risus sardonicus. The attendant states that she has to be held when seated on the night stool, or she would jerk herself off on to the floor. She died on December 11.

Autopsy (twenty-nine hours later).—Body poorly nourished; rigor mortis present. Skull neither thick or dense: basal vessels healthy. Meninges very thin and transparent, stript readily. Brain very large and pale, with no atrophy or dilatation of lateral ventricles. Lungs small, pale, crepitant; no pleural adhesions. Left side of heart slightly hypertrophied, and mitral orifice small. Slight calcareous deposit at base of aortic valves. No atheroma of aorta beyond. Liver soft and pale. Spleen small and firm. Kidneys small, dark purple colour; cortex tough; capsules slightly adherent.

Weights in grammes.—Cerebrum (stript), 1,132; meninges, 18; cerebellum, 139; stem, 30; total, 1,321. Heart, 215; liver, 885; right kidney, 101; left, 107; spleen, 59.

No. 4.—M. E. B., female, admitted December 20, 1897, aged 37, single. First attack; has always been considered hysterical, but for the last year has been nervous, apprehensive, and could not be induced to leave the house. Gradually lost use of her legs and hands, and the latter shook so much that she could not hold a cup. At times would fall down. Took to her bed several weeks before admission, and a few days ago developed screaming attacks. Said to have been a sober woman.

On admission is ill and unable to walk; slightly bedsore. Temperature, 99·6°. Complexion coarse, skin greasy and blotchy. Knee-jerks absent, plantar reflexes absent, pupils equal. Has bronchitis. Her sensitiveness to pricking is *nil* in face, neck, chest, arms, legs, and there is no local reaction. Is very apprehensive, and shrinks away when one goes near her. Keeps ejaculating little phrases *à propos* of nothing, *e.g.*, "Who speaks English?" "Isn't Mr. Archer?" "For grief I shall read," "*Do* help me." Shouts occasionally. Is dirty in habits. To most questions she pays no heed, but when asked her name replied, "Why, Brass."

Remained in much the same state till she died on December 24, 1897.

Autopsy (three-and-a-half-hours later).—Fairly nourished. Skull heavy and dense; vessels healthy. Meninges congested, but not thick or adherent; they were clear. Some atrophy of the frontal convolutions. Lateral ventricles not dilated. Brain of normal consistency. Evidence of emphysema and bronchitis. Heart muscles soft, apex fatty, valves fairly healthy. Liver soft and dark coloured. Spleen soft. Kidneys fairly healthy.

Weights in grammes.—Cerebrum (stript), 950; meninges, 13; cerebellum, 143; stem, 31; total, 1,137. Heart, 289; liver, 1,629; spleen, 152; right kidney, 145; left kidney, 170.

Sections of the popliteal, post-tibial, radial, and median nerves were examined, after fixing in osmic acid. The peripheral nerves showed very marked changes, some bundles being nearly devoid of all myelin rings (radial and post-tibial). The more central nerves were affected, but to a much less extent.

No. 5.—A. E. C. This woman was first admitted on June 25, 1889, suffering from melancholia, and was discharged, recovered, August 21, 1889, and re-admitted May 13, 1890. Profoundly depressed, restless, and agitated, constantly ejaculating, "Oh, whatever *shall* I do!" in an agonised tone. She remained in this state of deep depression till her death, but up to her last illness she was clean in habits and capable of doing ward work,

and never showed any motor troubles. In February, 1898, her arterial tension was 210 mm. Hg. On January 24, 1899, she went to bed with a sore throat, which proved to be diphtheria, and she died February 6 following. Her temperature during this period was always subnormal (taken in the rectum). She was 69 years old.

Autopsy (five hours after death).—She had a thick, ash-grey membrane lining the whole of her œsophagus, but stopping at the cardiac orifice of the stomach; small infarct in spleen. Thoracic viscera fairly healthy. Kidneys granular. Liver soft and dark. Head: basal arteries atheromatous. Large collection of fluid under the meninges, which showed here and there small opaque patches, but were elsewhere thin and translucent, and not adherent. Atrophy of convolutions most marked in parietal regions. No marked dilatation of ventricles.

Weights in grammes.—Cerebrum (stript), 1,084; meninges, 15; cerebellum, 147; stem, 35; total, 1,283. Heart, 238; liver, 1,102; spleen, 129; right kidney, 94; left, 111.

This case is out of harmony with the others, inasmuch as she was not demented in the ordinary sense of the word, and did not exhibit any of the motor troubles seen in all the other cases; but it has to be taken into consideration that she was confined to bed for nearly a fortnight prior to death with a malignant form of diphtheria, and the question arises whether the poison of the diphtheria was the cause of the changes found in the nerve-cells, or whether these changes were associated with her prolonged and profound melancholia.

I cannot help thinking that having regard to the unanimity with which motor disturbances were noted in all the other cases with similarly affected cells, the changes noted in her case were the result in part at least of the diphtheria, although in another case examined shortly before I was unable to discover any notable alteration in the nerve-cells of similar regions.

In the case under discussion, however, the form of disease was asthenic with a persistent low temperature, and in the latter it was rapidly fatal with a considerable pyrexia (104·6).

No. 6.—Male, aged 36. Admitted August 17, 1899. Father insane. First attack of recent origin. The facts certifying to his insanity were incoherence, violence, raving. On admission he weighed 138 lbs., and was in fair condition. Temperature normal. Knee-jerks present. Tongue furred. Pupils equal. He was very restless and incoherent, and paid no heed to questions. Threw himself into peculiar attitudes owing to involuntary jerking of

FIG. 1. ($\frac{1}{12}$ oil, No. 2 eye-piece, Beck.)—Cell with nucleus distorted and bulging from the side of cell body. A darkly stained cell body border. Some yellow pigment at right edge. A darkly stained irregular border separates the pale apex from the cell body. In the former are a few very fine short chromophile threads.

FIG. 2. ($\frac{1}{12}$ oil, No. 2 eye-piece, Beck.)—In this cell the whole of the centre is occupied by a pale washed-out hyaline looking mass. No nucleus is seen, but in serial sections one is seen to be present, situated at the upper right hand side of cell, about where the darker area is shown in the photograph. A mass of rather coarsely grained pigment is seen at the lower left hand border of cell.

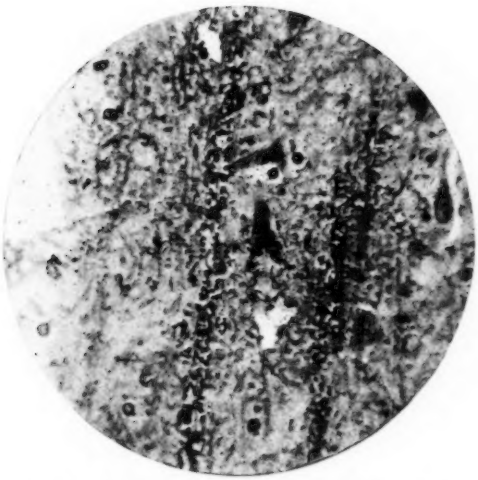

FIG. 3. ($\frac{1}{4}$, No. 2, Beck.)—A degenerated myelin nerve fibre seen in centre of figure, others to the right. The axis-cylinder can be seen in places.

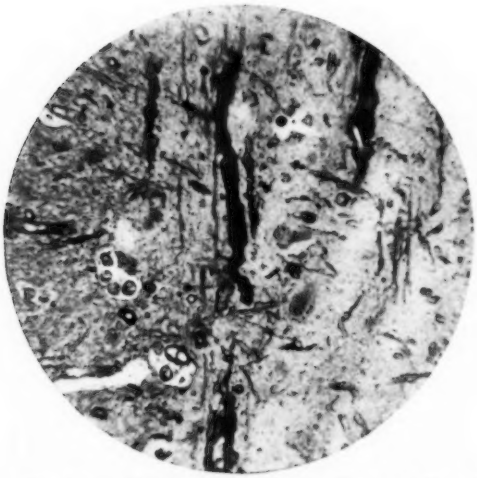

FIG. 4. (Same magnification.)—Normal myelin nerve fibres from other parts of same section.

the limbs, and had to be put in the padded room to prevent him from injuring himself, as he could not stand. Refused food. Rapidly got weaker, but still kept restless and very resistive; laughed causelessly, was dirty in habits. His temperature was subnormal on the two occasions it was taken. Became collapsed on the 21st, and died August 22, 1899.

Autopsy (a few hours later).—Beyond acute congestion of lungs the thoracic and abdominal viscera appeared healthy. Head: skull thick, basal vessels healthy. Meninges very opaque over the vertex, thick, not adherent. Excess of subarachnoidal fluid. Atrophy of convolutions over vertex. Slight dilatation of lateral ventricles. Brain of normal consistency. Cerebellum large. The meninges over the superior worm were opaque and thickened.

Weights in grammes.—Cerebrum (stript), 1,169; meninges, 28; cerebellum, 179; stem, 36; total, 1,412. Heart, 329; liver, 1,650; spleen, 85; right kidney, 111; left, 121.

No. 7.—W. A., male. Admitted July 2, 1892. Aged 52. Father melancholic and committed suicide. This was his first attack. He was acutely melancholic—the picture of misery—and gradually passed from this state into dementia. Picked his hands and head. Paresis of lower limbs was noted about two years after admission. He was bedridden for over a year, and died in March, 1898, of phthisis.

Autopsy (sixteen-and-a-half hours after death).—The lungs showed extensive tubercular changes. There was some atheroma of the aorta. Liver soft and pale. Kidneys pale and tough. Capsules stript well. Head: the basal vessels were atheromatous. The meninges were thick and opaque, but stript readily. Some atrophy of frontal convolutions. Lateral ventricles much dilated. Brain pale and firm. Cerebellum firm.

Weights in grammes.—Cerebrum (stript). 1,084; meninges, 24; cerebellum, 154; stem, 34; total, 1,296. Heart, 214; liver, 1,028; spleen, 109; right kidney, 101; left, 105.

OBSERVATIONS ON THE ASCENDING TRACTS IN THE SPINAL CORD OF THE HUMAN SUBJECT.

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THIS note deals with the following ascending tracts in the human spinal cord.

(1) Postero-median column.

(2) Dorsal ascending cerebellar tract.

(3) Ventral ascending cerebellar tract, including the nature of the fibres in the ventral lip.

(4) The intra-spinal portion of the fourth posterior cervical root.

The observations were made in two cases which were examined sufficiently early to allow the Marchi method of staining to be used. Such cases are comparatively rare, and more observations are necessary before the course of these tracts can be said to be thoroughly known. We are indebted to Sir William Banks and Mr. Rushton Parker, for permission to record this note.

In the autumn number of BRAIN, 1898, Bruce gave a careful and full account of similar observations in which also the Marchi method was available. With much of the description of this author we are in agreement, but some interesting points not shown in his figures will be described in this paper. A contribution on the same subject has recently been published by Risien Russell.[1]

[1] BRAIN, Summer, 1898.

The first case was one of caries of the spine, in which microscopical examination showed that at the level of the lesion in the mid-thoracic region, the cord was completely disintegrated.

The other case was a severe crush, the cord being entirely destroyed at the level of the tenth dorsal segment, with extensive consequent myelitis. There was also severe damage to the fourth posterior cervical root on the left side.

The tissues after hardening in Müller's fluid were stained by the Marchi method and sections cut after paraffin embedding.

(I.) *Postero-median column.*—The postero-median column, including the septo-marginal tract of Bruce and Muir was completely degenerated as high as the funiculus gracilis. It ended entirely in this nucleus. In the lowest cervical

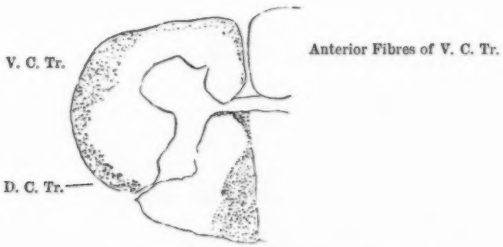

FIG. 1.
Spinal cord at the fifth cervical segment.

region the degeneration extended markedly up to the posterior commissure and spread along it as far as the margin of the posterior horn, thus embracing the cornu-commissural zone of Marie (fig. 1).

At the level of the fourth cervical root this appearance was much less marked (fig. 2), and just below the decussation (fig. 3) the area had assumed a distinct wedge shape, this feature being in accordance with Singer's law.

(II.) *Dorsal cerebellar tract.*—The course of this tract in the spinal cord is well seen in figs. 1, 2, 3. In this situation it has the position usually described. Sections in

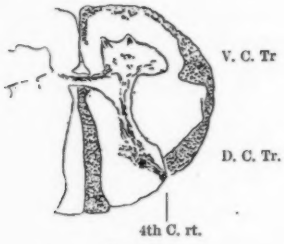

FIG. 2.
Spinal cord at the fourth cervical segment.

the lower cervical regions show that it is closely applied to the posterior horn, spreading inwards along its outer border for a considerable distance. In the lower medullary region

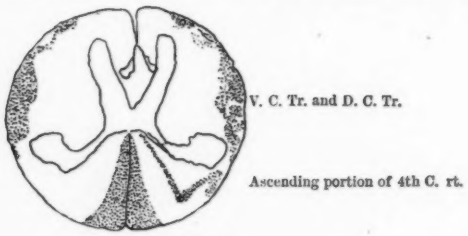

FIG. 3.
Spinal cord at the lower limit of the pyramidal decussation.

(figs. 4 and 5) it forms, together with the ventral cerebellar tract a well defined wedge-shaped area just anterior to the substantia gelatinosa of the fifth nerve.

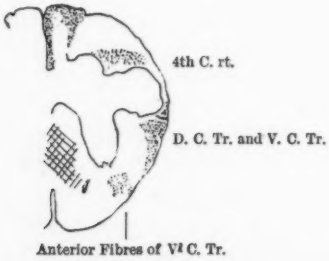

FIG. 4.
Medulla below the olivary body.

In our sections a distinct peripheral band of degenerated fibres is seen to connect this area with the degenerated fibres of the funiculus gracilis. We have not seen such a

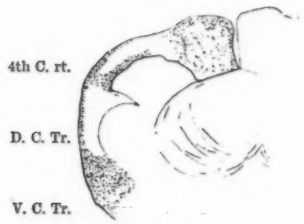

FIG. 5.
Medulla at the lower limit of the inferior olivary body.

condition described elsewhere, and are in doubt whether to consider all these degenerated fibres as part of the dorsal cerebellar tract.

In fig. 6 the tract occupies the central part of the corpus restiforme in a position described by other authors, and in fig. 7 it is seen passing into the cerebellum. We regret that we have been unable to trace its further destination.

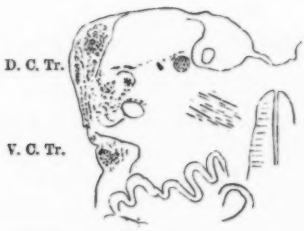

FIG. 6.
Medulla at the middle of the olives.

(III.) *Ventral cerebellar tract.* — The position of this tract in the spinal cord is shown in figs. 1, 2, 3. The main portion of the fibres form a wedge-shaped area opposite to the lateral part of the anterior horn, extending close up

to the periphery. This peripheral situation differs from that given by Risien Russell,[2] but is in agreement with the description of other authors. A marked feature in our sections is the presence of a considerable number of degenerated fibres occupying the periphery of the most anterior part of the white matter, and dipping down along the anterior fissure.

Degenerated fibres in a similar position have been noted by Sherrington[3] in the monkey after complete transection of the cord.

This condition in the human cord does not appear to have been noted by other writers. Tracing these fibres upwards it is seen that the formation of the pyramids pushes them

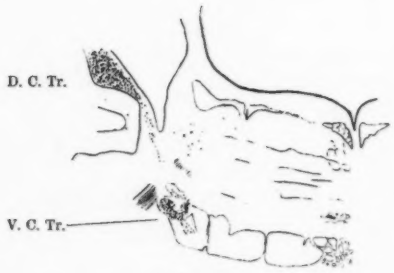

FIG. 7.
Pons at the exit of the fifth nerve.

backwards. At this level (fig. 4) the greater part of the fibres pass inwards and become dispersed in the white matter between the posterior border of the pyramid and the lateral nucleus of the medulla. Our sections do not give positive evidence whether these fibres terminate in the lateral nucleus or not, but such a condition appears probable.

In fig. 6 the main part of the ventral cerebellar tract is seen just external to the inferior olive. In fig. 7 it is placed more deeply, just ventral to the outgoing fifth nerve. In fig. 8 it is seen curving round the superior peduncle of

[1] *Journ. of Physiology*, vol. xiv., 4 and 5.
[2] *Loc. cit.*

the cerebellum, and entering the superior medullary velum in an identical position to that described by Bruce. A few degenerated fibres were found at a slightly higher level, but the material at our disposal unfortunately did not allow of our determining whether these passed to the inferior corpora quadrigemina.

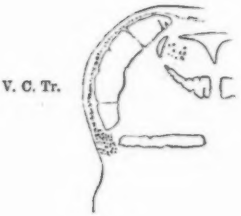

FIG. 8.
Pons at its upper limit.

(IV.) *Spinal portion of the fourth posterior cervical root.*—In one of the preceding cases this root was considerably damaged, and by means of the Marchi method its course could be accurately traced.

(*a*) *Descending portion.*—This is found to occupy an elongated zone in the middle of the postero-external column. It is well marked in the middle of the fifth segment, and scattered fibres can be traced as far as the lower part of the sixth segment. Throughout their descending course these fibres give off collaterals to the posterior horn.

(*b*) *Ascending portion.*—Fig. 2, which is at the level of the injured root, shows the entrance of degenerated fibres into the postero-external column, and the rich distribution of the reflex collaterals of Kölliker, which appear to be chiefly directed towards the postero-lateral group of cells. Other evidence tends to show that this group of cells receives special innervation from the posterior afferent root fibres. The collaterals are confined to the same side as the root lesion, and although more numerous in the fourth segment, are found in the segment above, and also, as previously stated, in the fifth segment.

Fig. 2 further shows a marked degeneration in the anterior commissure. The fibres can be traced from the anterior horn on the side of the lesion to the white matter between the fissure and anterior horn of the opposite side.

This decussating system of degenerated fibres was limited to the region of the injured root. In sections higher up it was not possible to distinguish between these fibres and those previously noted, which are in relation to the ventral cerebellar tract. Under the name of "crossed afferent tract" Edinger describes in this position a system of fibres, whose origin is from the posterior roots, ascending to terminate in the corpora quadrigemina or optic thalamus. Observers (Mott and Langley) who have noted this tract in experimental cases are divided in their opinion as to whether the fibres take origin in the posterior roots or in the most dorsal segment of the posterior cornu.

In our case there was no indication that the posterior cornu was injured, and hence this observation tends to confirm Edinger's view.

Sections (fig. 2) at the site of the injured nerve also showed distinct degeneration in some fibres passing from the anterior horn, which were limited to the side of the lesion. These fibres were too numerous to be neglected, but it did not appear that they were continuous with those entering the posterior horn. We are therefore unable to give a definite opinion as to their precise meaning.

The ascending portion of the fourth root in the third and higher segments occupies (fig. 3) an elongated area in the median part of the postero-external column, extending ventrally as a narrow strip to the base of the posterior horn, while dorsally the area widens and curves outwards towards the posterior root zone. In figs. 4 and 5 the fibres are seen collected around the external cuneate nucleus in which they end.

A STATISTICAL INQUIRY INTO THE PREVALENCE OF EPILEPSY AND ITS RELATION TO OTHER DISEASES.

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THE following paper is based on an inquiry into the occurrence of epilepsy or epileptiform convulsions in the family or personal history of 5,000 patients, who at the time of the inquiry were complaining of other than epileptic disease. From the results so obtained I have endeavoured to ascertain what, for want of a better term, may be called the average accidental incidence of epilepsy in family and personal history, and thus establish a standard from which the part, if any, played by epilepsy in the predisposition to other diseases may be decided.

The term "accidental" must not be accepted too literally. There are certain diseases, such as insanity, chorea, and some chronic nervous diseases, with the liability to which an epileptic inheritance or the occurrence of past personal epileptic fits has an established relation. In these cases, therefore, the patients will give a personal or family history of epilepsy in numbers altogether in excess of those met with in the case of such disorders, for example, as the specific fevers or diseases of the skin, with which epilepsy has apparently no relation at all. Nevertheless, a certain small proportion of the latter patients will give an epileptic history, owing to the fact that epileptics and those inheriting a family tendency to epilepsy are at least as liable to contract such disorders as other more healthy individuals,

and it is to express this fact that I have used the term accidental. Yet I do not intend to assert that the relationship between epilepsy and these disorders is necessarily of an entirely accidental character, for it is always possible that the epileptic taint may be one of a long series of links in the chain of causation. Indeed, my figures give some support to this idea.

Besides perhaps throwing some additional light on the natural history of epilepsy, there are certain disputed points which can only be decided by such an inquiry. For example, it is still a moot point whether phthisis and epilepsy have any special relationship with one another. Sir William Gowers obtained a phthisical family history in 39 per cent. of 200 epileptic patients, but came to the conclusion that the frequency of this association might probably be accounted for by the wide-spread diffusion of phthisis; whilst Dr. F. Roberts, who at his request investigated the matter from the other point of view, found that an epileptic family history was only forthcoming in 2·5 per cent. of his phthisical cases, a percentage which he thinks is not above the average. But these figures are inconclusive owing to the lack of any standard with which they can be compared, and which gives the incidence of phthisis and epilepsy in other cases of disease. A knowledge of the average occurrence of epilepsy, which it is the object of this paper to establish, will, of course, determine only one side of these and similar questions; but I have endeavoured to obtain some statistics as to the prevalence of phthisis also, and thus to decide the relationship of that disease to epilepsy from both points of view.

Method of the Inquiry.

The 5,000 cases on which the epileptic statistics are based were obtained entirely from my own out-patients. They therefore all belong to the same class of society, and are practically consecutive. Each patient on his first attendance was asked as to the existence of "fits"

in his family or personal history, and the case with its result entered under the corresponding diagnosis. It is of course true that in the hurry of a crowded out-patient department diagnosis is often of a somewhat rough description. Nevertheless, I have taken the greatest possible care to obtain accuracy, and have freely revised the diagnoses as occasion has demanded. Moreover, whenever the diagnosis has seemed to be doubtful, I have excluded the case from any of the definite groups and have placed it in a separate list reserved for cases of a doubtful nature. I therefore venture to believe that no very serious error has crept into the results from this source.

The inquiry for the occurrence of "fits" has necessarily elicited a history of hysterical as well as epileptic convulsions. My table therefore includes both varieties. It is often, however, a most difficult matter to distinguish between epileptic and hysterical fits from the account of an uneducated person, and in no less than 20 per cent. of all the fits of which a history has been given, I have been entirely unable to decide to which category they should belong. In these cases, therefore, the fits have been placed in a separate group under the heading "Doubtful." In the remaining 80 per cent., in which I have arrived at a conclusion one way or the other, I believe that I have had good grounds for my decision.

The points on which I have relied in attempting to distinguish between the two sets of convulsions are those usually accepted. Thus, I have taken as fairly conclusive of epilepsy, tongue-biting, passage of urine or motions in the fits, and occurrence of serious bodily mischief, such as bad burns or scalds, and really bad falls, &c., though I am, of course, aware that such accidents are not absolutely conclusive against hysteria. Detention in a lunatic asylum has also as a rule been taken as evidence that the fits were of an epileptic character. Again, in not a few cases my informant has given me so good an imitation of the movements of an epileptic fit as to convince me of its nature. On the other hand, screaming, fighting and struggling, talking, crying, and banging the head, have been accepted,

with other points, as evidence of hystero-epilepsy. Leading questions have in all cases been as far as possible avoided.

In some cases there has seemed to be a fairly clear history of the occurrence at different times of both varieties of fits. This is only in accordance with the well-known tendency of the two conditions to occur in association with one another.

Whatever pains be taken, however, in the examination of the point, some mistakes in interpretation will probably occur. In order to minimise the difficulty as far as possible, I have given in the two final columns of the table, first, the total number of personal fits of all sorts (epileptic, hysterical and doubtful added together), and secondly, the sum total of all the fit histories, family as well as personal, hysterical and doubtful as well as epileptic. In these columns, at any rate, there is no room for fallacy.

No notice has been taken of any fits occurring in the descendants of the patients, owing to the possibility of the inheritance arising from the other parent. Moreover, no fits occurring in individuals under three years of age have been registered, nor any for which the action of a poison or source of irritation might have been responsible. The personal fits of patients suffering from Bright's disease, lead poisoning, worms, &c., have therefore been omitted, unless they occurred at a period previous to the exposure to the poison or irritation.

Finally, it must be remembered that the figures undoubtedly understate the incidence of both varieties of fits, owing to the very imperfect knowledge of family history prevalent amongst the working classes. Both epileptic and hysterical inheritance must therefore be more frequent than is shown by my figures.

Arrangement of Statistics.

The cases are arranged under twenty-one different diagnoses, one additional group being added under the heading "Various." The latter contains all the disorders the representatives of which have not amounted to seventy-

five in all, this being taken as the smallest number which would justify separate analysis. Another group originally contained cases of doubtful diagnosis, but this has now been added to the "Various" cases.

Some of the groups need a brief explanation :—

Anæmia.—This group is made up entirely of young women, mostly from 15 to 25 years of age. All special forms of anæmia, with the exception of chlorosis, have been placed in the "Various" group.

Bright's disease.—The different varieties of Bright's disease have all been classed together, seeing that their separate analysis has given closely similar results.

Dyspepsia.—The dyspeptic group contains undoubtedly a very miscellaneous set of cases. I have, however, endeavoured to simplify it by separating the male from the female cases, holding that the latter constitute a far more neurotic group. It is still the case that very various forms of stomach upset are included under the same heading, but it would be somewhat hazardous to attempt much subdivision.

Debility.—The patients here consist largely of the over-worked and underfed men and women who are met with constantly in hospital practice in large manufacturing towns. They were suffering from no ailment which would place them in any of the other groups.

Headache.—No patient has been included in this class in whose case a definite cause could be assigned for the complaint. The cases are those of so-called functional headache.

Nil.—In this group have been placed those patients with whom a careful examination has convinced me that there has been little or nothing the matter.

Rheumatoid arthritis.—Under this head a few cases of chronic "rheumatism" other than rheumatoid arthritis have probably been included.

Various.—As already explained, this class contains the diseases the representatives of which have not amounted to seventy-five in number, and also cases of doubtful diagnosis.

Results Arrived at.

The table of results is as follows :—

	Cases.	Ep. Fam. Hist. Per cent.	Ep. Personal. Per cent.	Ep. Fam. and Personal. Per cent.	Hyst. Personal. Per cent.	Hyst. Fam. and Personal. Per cent.	Doubtful. Per cent.	Total Personal. Per cent.	Total Fits. Per cent.
Anæmia	431	4·4	0·9	5·1	2·8	8	2·6	5·1	14·6
Bright's Disease	136	2·2	1·5	3	0·7	3	2·2	3	8·8
Acute Bronchitis	137	4·4	4·4	7·3	0·7	4·3	2·2	5·8	13·9
Chronic „	211	3·3	2·8	4·7	0	0·5	2·4	3·3	7·5
Chorea	146	17·8	6·2	21·2	1·4	6·2	2·1	7·5	25·3
Dyspepsia (Male)	130	1·5	0	1·5	0	4·6	2·3	0·8	8·4
„ (Female) ..	375	5·1	1·6	6·7	2·4	6·9	1·6	4	13·3
Debility	183	5·6	1·1	5·6	0·6	3·9	2·8	2·2	11·7
Fevers	114	3·5	0	3·5	0·9	3·5	0·9	1·8	7·9
Headache	131	10·7	3·1	13	1·5	3·8	4·6	7·6	20·6
Heart (Valvular)	169	4·7	4·1	8·3	0	4·1	0·6	5·3	13
„ (Muscular)	77	5·2	1·3	6·5	1·3	2·6	0	2·6	7·8
Myalgia	123	7·3	0	7·3	0·8	4·9	2·4	3·3	13·8
Neuritis	75	6·7	0	6·7	0	2·7	2·7	1·3	10·7
Neuroses	321	5·9	1·9	7·5	2·8	7·2	1·9	5	15·3
Chronic Nervous Diseases	111	9	4·5	11·7	0·9	2·7	1·8	5·4	14·4
Nil	136	2·9	0	2·9	0	0·7	0·7	0	4·4
Phthisis and Chr. Pneum.	276	6·2	4·3	10·1	0·7	3·3	0·7	4·7	13·8
Acute and Subacute Rheumatism	132	4·6	2·3	6·1	2·3	4·6	2·3	5·3	12·1
Rheumatoid Arthritis ..	87	8	1·1	8	0	0	2·3	3·4	10·3
Skin Diseases	174	3·4	0·6	4	1·1	4·6	2·3	2·3	10·3
Tonsillitis, Pharyngitis and Stomatitis	127	4·7	1·6	4·7	1·6	3·9	0·8	3·1	7·9
Various	1198	4·2	1·1	5·2	0·8	3·2	1·8	2·3	10
Total (Average)	5000	5·2	1·8	6·6	1·1	4·2	1·9	3·6	11·9

Note.—In order to save multiplication of columns, Family History includes patient's own generation as well as preceding generations.

The chief point of interest of the average thus obtained lies in the frequency with which it shows that fits of one sort or another occur amongst the working classes. Thus, no less than 11·9 per cent. of my out-patients, excluding those attending directly for epilepsy or hystero-epilepsy, gave me a family or personal history of fits, whilst 3·6 per cent. had suffered from fits themselves at some period of their lives; 5·2 per cent. gave a family history of epilepsy,

and 1·8 per cent. had themselves had epileptiform convulsions. It has already been noted that the figures understate the case as far as family history is concerned.

The average is inaccurate, however, if it be taken to represent the extent to which personal and family histories of epilepsy and hystero-epilepsy are to be found amongst persons who are suffering from complaints with which these disorders have no recognised relation; for the cases on which the epileptic average is based include both chorea and the chronic nervous diseases, and the hystero-epileptic average is similarly invalidated by the inclusion of anæmia, the neuroses, and the female cases of dyspepsia, with all of which hysteria may be accepted as possessing a definite relation. Headache, again, has an easily understood relation to both epilepsy and hystero-epilepsy; whilst finally it is known that epileptiform convulsions are not uncommon complications of valvular heart disease. Hence, the first step towards obtaining the true "accidental" incidence of epilepsy and hystero-epilepsy must be to subtract all the above-mentioned conditions, and to take the average of those remaining. The average now stands as follows, the items being arranged in the same order as that adopted in the table:—

3306	4·4	1·5	5·6	0·7	3·2	1·8	2·8	10

I do not, however, think that even this represents accurately the average at which I wish to arrive. On studying the table certain diseases are seen to stand out, as regards the incidence of epilepsy distinctly above the corresponding figures in the last obtained average. These diseases are phthisis and chronic pneumonia, and acute bronchitis, as regards epileptic personal history, and acute and subacute rheumatism as regards the total number of personal fits of all sorts. To these I should also add rheumatoid arthritis as regards epileptic family inheritance, were it not that there is a complete absence here of any record of hysterical fits, and that the total fit incidence is scarcely above the average. These facts suggest that a larger number of cases would probably level the average

by the inclusion of cases of hystero-epilepsy. The group of myalgias also claims removal on the ground that it exceeds the average as regards both family epileptic inheritance and the total fit incidence; but, although the connection of these cases with rheumatism might possibly justify me in dealing with them in the same way as the above mentioned diseases, the group is perhaps somewhat ill-defined, and is, therefore, better retained.

At the risk of appearing to assume the points under discussion, I yet think that the cases of phthisis, chronic pneumonia, acute bronchitis, and acute and subacute rheumatism must be withdrawn in order to arrive at the final accidental incidence of epilepsy; for, if my figures be correct, they afford some evidence that these conditions stand to it in some special relation. In other words, they are now under suspicion. If they have no such special relation, their removal should not affect the results; and if, on the other hand, they do stand in some special relation, their retention in the table clearly vitiates the average. The final "accidental average" therefore now stands as follows:—

Cases.	Ep. Fam. Hist. Per cent.	Ep. Personal. Per cent.	Ep. Fam. and Personal. Per cent.	Hyst. Personal. Per cent.	Hist. Fam. and Personal. Per cent.	Doubtful. Per cent.	Total Personal. Per cent.	Total Fits. Per cent.
2768	4·3	1	4·9	0·7	3·1	1·9	2·3	9·5

These figures give, I think, the "accidental" incidence of epileptic and hysterical convulsions as nearly as it can be arrived at.

Provisional Inferences.

On comparing this average with the figures in the table, the following points are to be noted:—

Anæmia.—The epileptic incidence is almost identical with the average, but the personal and total incidence of hysterical convulsions show a considerable increase—namely, 2·8 and 8 against 0·7 and 3·1 per cent. respectively.

Acute bronchitis.—The personal epileptic fits stand at 4·4 instead of 1 per cent. The percentage of personal fits, all sorts included, is 5·8 as compared with the average of 2·3.

Chronic bronchitis.—In contra-distinction to the total fit incidence of the cases of acute bronchitis (13·9 per cent.), the total liability is here below the average (7·5 instead of 9·5 per cent.). It is however to be remarked that even here the average of personal epileptic fits is nearly three times the normal (2·8 as compared with 1), whilst the total personal fits stands at 3·3 instead of 2·3 per cent.

Chorea gives the largest epileptic incidence, and shows some increase also in hysterical relationship. The total incidence is 25·3 instead of the average 9·6 per cent.

Headache gives the next largest percentage of fit incidence of all classes. The family epileptic percentage is 10·7 compared with the normal 4·3; the personal epileptic incidence is 3·1 instead of 1 per cent. Taking fits of all sorts together, the personal percentage is 7·6 compared with 2.3, and the total 20·6 instead of 9·5.

Heart (valvular.)—The figures here obtained support the view that patients suffering from cardiac valvular disease are more subject to the occurrence of eleptiform fits than other people. Thus, personal epileptic fits were noted in 4·7 per cent. of the patients, as compared with the average of 1 per cent.; and in 2·4 per cent. there was good reason for thinking that the fits had occurred after the onset of the endocarditis. In 1·2 per cent., however, this was certainly not the case; whilst in the remaining 1·2 per cent. there is no evidence on the point one way or the other.

Neuritis.—The only point to be noticed in this group is a moderate increase in the epileptic family inheritance.

Neuroses show an increase in the total personal fits (5 per cent. instead of 2·3), whilst the percentage of fits of all sorts combined is 15·3 instead of 9·5. The increase is here chiefly on the hysterical side, though there is also some excess of epileptic inheritance.

Phthisis and chronic pneumonia give a moderate increase in epileptic family history (chiefly, however, in the patient's

own generation), and a marked increase in the number of personal epileptic fits (4·3 instead of 1 per cent.). Taking the personal fits of all sorts and the total combined fits, the numbers are 4·7 and 13·8 as compared with the normal of 2·3 and 9·6 respectively.

Acute and subacute rheumatism.—There is again a marked increase in the total number of personal fits, those due to epilepsy and hysteria being now equal in number. But I have unfortunately omitted to note the condition of the heart in many of these cases, and the part played by this factor is therefore uncertain. The relation of rheumatism thus needs further investigation.

Chronic nervous diseases.—It is unfortunately useless to split this group up into the separate diseases of which it is composed, for the number of cases in each category is too small for individual analysis. There is, as would be expected, a great increase in both family and personal epileptic incidence.

Speaking provisionally—for some of the suggestions need the support of a larger series of cases—the foregoing points give some ground for thinking that the various disorders may be arranged in an ascending scale according to the degree to which the family and personal histories of the patients are tainted with epilepsy.

(1) It is to be noted that the group ("Nil") which includes the individuals nearest to a normal state of health, gives a far lower fit incidence than that of any of the other groups. This incidence amounts to but 4·4 per cent. for fits of all classes, no personal fits of any sort being registered. There appears, therefore, to be some reason for believing that an inquiry into the history of a perfectly healthy set of persons would give a uniform average of fit incidence much lower than that to which the name accidental has hitherto been applied. The "accidental average" must therefore be considered as applying to the sick members of the working population only.

(2) More than 50 per cent. of the more definitely ailing patients give a fairly uniform average of epileptic incidence —that, namely, established in my final average.

(3) A group including acute (? also chronic) bronchitis, phthisis, chronic pneumonia, valvular heart disease, and acute and subacute rheumatism, shows a striking increase in the number of patients who have themselves suffered from epileptiform convulsions.

(4) Finally, the most marked epileptic incidence is to be found in the cases of chorea, functional headache, and chronic nervous disease, in all of which the relationship reaches a high degree.

Cases of Lung Disease.

One of the chief points of interest in the above conclusions lies in the apparently large number of patients affected with phthisis, chronic pneumonia, and acute bronchitis, who have suffered from epileptiform convulsions. I therefore give a few further details concerning these cases.

A possible source of fallacy as regards the phthisical cases lies in the fact that they are practically exposed to an age limit, whereas the cases with which they are compared include patients of all ages. Thus, in the phthisical cases there are but very few patients below the age of 15, and it is clear that in order to bring the accidental average into strict comparison with the phthisical average, all patients under this age should also be omitted. This correction, however, only brings the accidental average of personal epileptic fits up to 1·1 instead of 1 per cent., whilst the percentage of total personal fits of all sorts is changed from 2·3 to 2·5 only. The removal of this fallacy therefore, does not materially affect the result. In the cases of bronchitis no such correction is needed.

Again, I have looked carefully into the history of the individual fits in the phthisical and bronchitic patients, in order to see how they compare from the point of view of permanancy with the personal fits of which a history has been obtained in the whole series of 5,000 cases. The result is as follows:—

Personal epileptic fits occurring amongst the 4,597 patients who were not suffering from either phthisis or bronchitis:—

Confirmed Epilepsy, Past or Present.	Recent only.	Former and Temporary.
67·1 per cent.	19·7 per cent.	13·1 per cent.

Personal epileptic fits occurring amongst the 137 cases of acute bronchitis :—

Confirmed Epilepsy, Past or Present.	Recent only.	Former and Temporary.
83·2 per cent.	16·6 per cent.	0

Personal epileptic fits occurring amongst the 265 cases of phthisis, &c. :—

Confirmed Epilepsy, Past or Present.	Recent only.	Former and Temporary.
69·3 per cent.	15·4 per cent.	15·4 per cent.

It is therefore clear that the proportion of cases of confirmed epilepsy is materially above the average in the bronchitic cases, and slightly above it in the cases of phthisis.

The cases are as yet too few for any certain conclusions to be drawn, but the inference is strongly suggested that those patients whose nervous constitution is a weak one—as shown by the inheritance of an epileptic tendency, and especially by the occurrence of epileptic fits in the patients themselves—are directly predisposed to the development of serious lung mischief. The point needs, however, to be supported by a larger number of cases.

Inheritance of Phthisis by Epileptic Patients.

The establishment of the average inheritance of phthisis by hospital patients is an extremely difficult matter, owing to the looseness with which the term consumption is used to and by the working classes. The following table, based upon an inquiry into this point in the case of 2,966 patients, must therefore be taken with considerable reserve, as representing but little more than the mere statements of the patients as to the existence in their families of a history of phthisis. In all cases supporting evidence, such as hæmoptysis, has been asked for, but in a large minority it has been necessary to take the statements without cor-

roboration. The arrangement of the table is the same as before :—

	Cases.	Phthisis Inherited. Per cent.		Cases.	Phthisis Inherited. Per cent.
Anæmia	249	23·3	Heart (Muscular) ..	46	17·4
Bright's Disease ..	96	9·4	Hystero-Epilepsy ..	17	46·5
Acute Bronchitis	67	25·4	Myalgia	68	14·7
Chronic Bronchitis	118	13·6	Neuritis	48	14·6
Chorea	78	26·9	Neuroses	165	15·1
Dyspepsia (Male)	85	4·7	*Nil*	94	21·3
Dyspepsia (Female)	218	16·1	Chronic Nervous Dis...	57	14
Debility	97	18·6	Phthisis	149	31·6
Epilepsy	114	27·1	Acute & Subac. Rheum.	75	16
Fevers	57	26·3	Rheumatoid Arthritis..	50	20
Headache	66	10	Skin Diseases	118	17·8
Heart (Valvular)..	96	17·4	Tonsilitis, &c.	75	13·3
			Various	680	17

Total	2,966 ..	18·1 per cent.
Total (excluding Phthisis)	2,817 ..	17·4 per cent.

According to this table, it would appear that epileptic patients give a large inheritance of phthisis (27·1 per cent.). But the great irregularity of the results deprive this fact of much of its value. Thus, the specific fevers give a scarcely smaller inheritance—namely, 26·3 per cent., though the small number of cases may perhaps to some extent explain the difficulty. As far as it goes, however, the table supports the view that an inheritance of phthisis predisposes to the occurrence of epilepsy. It may be added that a similar belief—namely, the connection between chorea and tuberculosis—receives support from this table.

The general conclusion thus seems to be in favour of the view that epilepsy and phthisis have some connection with one another from both points of view. This conclusion, as far as epileptic incidence in the histories of phthisical patients is concerned, is not in accordance with that arrived at by Dr. F. Roberts, and quoted at the beginning of my paper; for although this observer did not

inquire into the occurrence of epileptiform convulsions in the personal histories of his patients, he found an epileptic family inheritance in only 2·5 per cent., as compared with the percentage of 6·2 obtained amongst my own phthisical patients. I cannot explain the discrepancy, but it appears to me possible that the explanation may to some extent lie in the different conditions of life obtaining in London and in a large manufacturing town such as Birmingham, and perhaps in some difference in the class of society from which the patients are drawn in the two cases.

A CASE OF ACUTE MENINGO-MYELITIS.

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SUMMARY:—Man, aged 37, exposed to severe cold on March 22. Great pain in the back from March 24 to April 11. Retention of urine on April 11, and rapid loss of power in the legs two days later. Complete flaccid paraplegia with sensory paralysis and loss of knee-jerks. Unexpected death from cardiac failure on the eighteenth day of the disease. Suppurative external meningitis in the dorsal region. Intense and mostly patchy myelitis from D 3 to L 3. Pus in the muscles of the back. A small localised empyema. No vertebral disease.

The following are short notes of the case.

A previously strong and healthy man, aged 37, was paving in Leeds on March 22, 1898—a snowy and bitterly cold day. On March 24 there was severe pain in the back, intermitting and stabbing. The pain was worse when he straightened himself after stooping. He gave up his work on April 9 for Easter. During the holiday he walked about, but often had the pain in his back. There was no pain or tingling in the legs. On April 11 he was unable to pass his urine. A little urine dribbled away on the 12th. The distension of the bladder caused him much pain. The bowels were very constipated. On April 13 the patient was catheterised and the catheter was used on five occasions before his admission into the Leeds General Infirmary on the 15th. On the evening of the 13th he got out of bed and quite suddenly lost the use of his legs and fell to the ground. Sensation was said to have been lost at the same time. During the night fæces were passed unconsciously into the bed. I saw the patient on the 14th with Dr. Spong, of Leeds, and he was admitted into the Infirmary on the following day. There was a history of syphilis fifteen years previously—a chancre, but no secondary symptoms; treatment was continued for thirteen weeks.

On admission.—The lower extremities were helpless and he could only alter the position of the trunk with difficulty, chiefly owing to the pain in the back. There was retention of urine and the stools were passed unconsciously. Pain was complained of round the chest about six inches above the umbilicus. The pain was not continuous, but of a tearing character and most intense in the epigastric angle. Above this level there was a zone of hyperæsthesia, and below it loss of sensation. The *legs* were completely paralysed so that not even the toes could be moved. The muscles were fairly well nourished but flabby. They were not tender. The superficial and deep reflexes were absent. Sensation was almost completely lost; the patient could not feel the prick of a pin or tickling the soles, but he could just feel a sharp rub against the leg. Sensation to heat and cold was quite abolished. There were no trophic disturbances. The *arms* were unaffected. There was no undue prominence of the spine. There was much tenderness over the lower dorsal vertebræ, and pain was complained of when a deep breath was taken. The patient also complained of inability to cough up the expectoration which now and again accumulated. The examination of the chest as far as it was possible to make it gave negative results. The urine, specific gravity 1029, acid, contained mucus but no albumen. April 21.—The patient's condition is unchanged. The urine remains strongly acid. April 26.—Condition much as before. He sometimes feels a tingling in the legs, especially the right. The anæsthesia begins just below the distribution of the sixth intercostal nerve. The temperature rises in the evening. The patient was in much the same state until the early morning of the 28th, when he was seized with cardiac failure and died on the eighteenth day of the acute myelitis and the thirty-sixth day of the disease.

Post Mortem.

The brain weighed 42½ ozs., and no evidence of disease was found in it. The meninges were healthy.

Spinal cord.—In the middle and lower parts of the dorsal region there was dirty looking pus on the outer aspect of the dura mater. The pia mater appeared healthy. Parts of the cord were obviously softened. Only one incision was made into it, and that through the lower dorsal region, where there was some bulging. The cord was almost diffluent here. The muscles of the back were infiltrated

with pus, especially on the left side. A very small empyemal cavity containing offensive pus was found close to the vertebral column on the right side. The lungs were otherwise healthy. The bladder was large and its walls thin, but the mucous membrane healthy. There was no evidence of cystitis. The kidneys were large, somewhat engorged with blood, but otherwise healthy. There was no pus in the ureter or pelvis of the kidney. The heart, including the valves, was healthy. The spleen was a little enlarged. The liver was also slightly enlarged, and adhesions were found between it and the diaphragm. These organs were otherwise healthy. Cultures were prepared from the pus outside the dura mater, and also from a softened patch in the cord. The same micro-organism (a large diplococcus) was obtained from both these lesions. The bacteriological examination was unfortunately not carried further, as the micro-organism died in a few days.

The whole cord was hardened in formol-Müller, and was divided into its segments. Portions from each segment from D 3 to L 4 inclusive were embedded in paraffin, the other parts of the cord being sufficiently firm to cut without embedding. Later each segment was again divided into pieces measuring about 2 mm., and the situations of the lesions as then seen were carefully noted.

The naked eye appearances of the cut sections of the hardened cord will first be described, as the lesions stood out very prominently. The results thus obtained were carefully compared with the changes observed in microscopical preparations from all parts of the cord. It is, of course, not assumed that the lesions visible to the naked eye corresponded exactly to the morbid change as seen under the microscope; but there was a very general agreement between the two. The disease was certainly of the disseminated and patchy type in nearly all parts of the diseased cord, and healthy nervous tissue was found between the patches. Sometimes these patches were very sharply defined from the healthy tissue as in D 4, but at other times the one faded more gradually into the other.

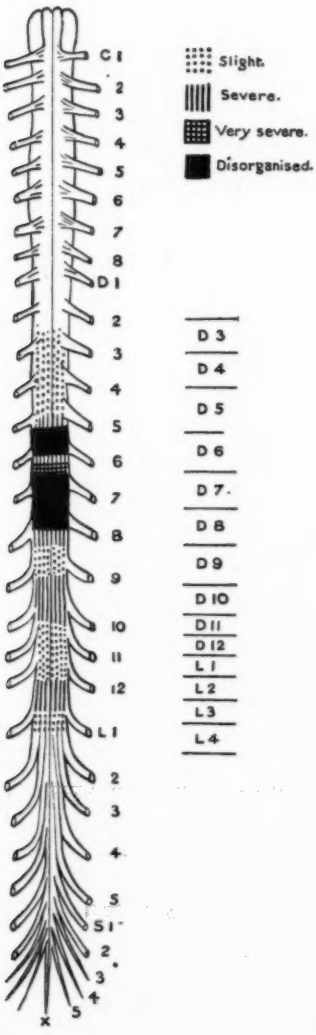
In its *longitudinal* distribution the disease was traced from D 3 as low down as L 3. The most intense disease was found in the lower part of D 5 and upper part of D 6, and again throughout D 7 and in the upper part of D 8. Here no distinct structure could be recognised by the naked eye, and an advanced disorganisation of the cord was made out by the microscope. Above D 3 there was an ascending degeneration, but no descending lesion could be ascertained in that part of the cord below L 3.

In its *transverse* distribution the disease was distinctly patchy, except where marked disorganisation was present. These patches were more numerous in the upper part of D 6, where in some places as many as nine patches could be counted in the cross section of the cord. In D 4 and 3 the disease was represented by a single, well-defined focus at the junction of the right anterior and posterior horns. Towards the lower limit of the lesion in the lumbar cord the patches were almost exclusively confined to the posterior columns or immediately adjacent parts. These patches were, generally speaking, much more abundant in the periphery of the cord than in the central parts, that is, they were placed in the peripheral circulation. At the upper limit of the lesion the patches were more centrally placed.

No distinct naked eye lesion could be made out in the hardened medulla oblongata, pons, cerebellum or cerebrum.

As regards the microscopical appearances, it must, of course, be borne in mind that the case terminated on the eighteenth day. The most marked changes were certainly centred in the parenchymatous elements.

There has been a great destruction of ganglion cells throughout the diseased portions of the cord; in fact, very few healthy ones are to be seen. Even in the lower lumbar segments they are considerably involved. Here the cells have lost their processes—have become rounded and translucent-looking, and the nucleus and nucleolus have disappeared. Neither vacuolation or pigmentation is seen. The nerve-fibres, including the axis-cylinders, are greatly altered in the diseased parts. Sometimes the axis-cylinder is so swollen that only a thin layer of


C 1
2
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D 1
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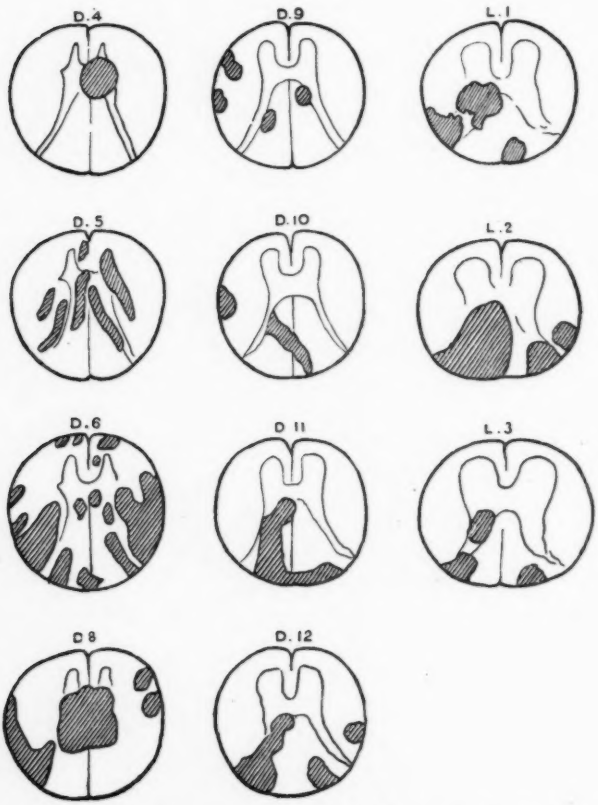
11
12
L 1
2
3
4
5
S 1
2
3
4
5
Slight.
Severe.
Very severe.
Disorganised.
D 3
D 4
D 5
D 6
D 7.
D 8
D 9
D 10
D 11
D 12
L 1
L 2
L 3
L 4

altered myelin surrounds it. At other times the breaking up of the myelin sheath is very obvious. In some places the nerve-fibres have largely perished, leaving hardly anything more than the glia network. The alteration in the



Ascending degeneration in the dorsal and cervical regions. A single small focus of disease in D. 3. D. 7 disorganised. L. 4 healthy.

interstitial tissue, including the blood-vessels, is of much interest, considering the undoubtedly infective origin of the disease. Vascular engorgement was not very marked in this

case. The vessels were, however, here and there crowded with blood-cells, and appeared then to be more numerous than usual (D 6). Very few hæmorrhages were visible; a large one was found in D 10 in the grey matter. No very distinct changes were to be made out in the vessel wall, and none in the perivascular sheath. Hardly any perivascular or other more general infiltration was seen. Thus the changes in the vessels and perivascular tissues are slight.

The changes in the connective tissue, including the neuroglia, were also not marked.

The bands of connective tissue passing into the cord from the periphery are more distinct than usual, and distended blood-vessels may at times be seen in them. There was very little overgrowth of the neuroglia, and in only a few places were spider-cells present in any abundance. Granules were only seen here and there. Large cells with small nuclei, staining by Weigert and other similar methods, were seldom found. Only a few corpora amylacea were present.

It would certainly appear that the stress of the disease, in this case, had fallen on the parenchymatous elements of the cord. If the pathological changes found were due to an infection or intoxication, then the vessels by which the disease-producing agent arrived should show changes in or about their walls; but such changes were only slight in this case. Nor was the lesion limited to any vascular territory, such as would suggest the deleterious action of some poison brought to it by the blood. Although, as mentioned above, the patches were often more abundant in the region of the peripheral blood supply to the cord, they were by no means limited to it.

The bacteriological examination made in this case was far too incomplete to allow of any conclusions being based upon it. Neither the pus of the empyema nor that in the muscles of the back was examined bacteriologically, but the resemblance in the naked-eye appearances of the pus in these two situations was considerable.

It is somewhat difficult to explain the exact relationship between the various lesions found after death, viz., the acute myelitis, external meningitis (without vertebral disease), the

small empyema, and the diffuse suppuration in the muscles of the back; but the infective character of these lesions cannot be gainsaid. It is thus difficult to say whether the suppuration outside the vertebral column was primary and the suppurative meningitis secondary, or whether, on the other hand, the suppurative meningitis was primary and the adjacent suppurations secondary. In the allusion to this subject in his well-known text-book, Dr. Gowers recognises the greater probability of the second relationship.

The case illustrates how the disseminated and transverse form of acute myelitis pass into one another. In some parts of the cord there was a total transverse lesion, whereas in other parts the patchy character of the disease was obvious enough.

Finally the symptoms noted during life corresponded very fairly well to the lesions found after death. The intense pain in the back must be largely, if not entirely, attributed to the external meningitis. That all functions dependent on the lumbar and lower dorsal cord were in abeyance is not to be wondered at, considering the intensity and amount of the disease present. The lesion in the cord extended rather higher than the symptoms indicated, but this was no doubt due to the fading off of the disease in a gradual manner from below upwards. Perhaps the unexpected and rapid death was due to minute lesions present in the medulla oblongata and pons. Thus in the region of the nucleus of the tenth nerve very small hæmorrhages were present, but this part of the examination was not as complete as it might have been. Under the circumstances it was almost impossible to recognise the small empyema and the suppuration in the back muscles during life, no doubt chiefly because they were not expected.

FRIEDREICH'S ATAXIA WITH KNEE-JERK SAND ANKLE CLONUS.

BY HOWARD GLADSTONE, M.D., C.M.EDIN.

Late House Physician to the Bradford Royal Infirmary.

In February, 1898, Arthur R., a lad of 17, was admitted to the Bradford Royal Infirmary from the out-patient room, suffering from spinal curvature and difficulty in walking. After examination he was found to present all the characters of a case of Friedreich's ataxia. The family history being investigated it was found that his eldest brother, William, was suffering from deformity of the spine, and was unable to stand. He was written to, and a few days later came into hospital with his brother, where they were both under observation for two months, under the care of Dr. James Kerr, who has kindly allowed me to record their cases.

It is the elder brother's condition which is referred to in the title, for it was found that, while presenting most of the classical features of the disease in a pronounced way, there was ankle clonus on the left side and retention of knee-jerks.

Ankle clonus has not been noted as a sign in any case of Friedreich's ataxia so far recorded, and naturally excites suspicion as to the correctness of the diagnosis. But his brother's case will be recognised as typical, and his own in other respects as strikingly characteristic of Friedreich's disease.

(1) William R., stocking-knitter, aged 24 years, the elder patient, was admitted February 23, 1899.

History.—Symptoms began at 16 years of age, with alteration in the shape of his feet, the instep became high and he could not lace up his boots. At this time he was beginning to grow rapidly. A year later he was taken to the Infirmary, on account of spinal curvature, for which plaster-of-Paris jackets were applied; the deformity nevertheless continued to progress until he ceased growing. His work was trying, for he was obliged to stand all

day long in the mill. From 17 years of age he experienced occasionally short stabbing pains in the shin bones. It was not until 18 years of age that he noticed anything wrong with his legs, but then walking became difficult. The difficulty slowly increased, so that fifteen months ago he had to stay at home; he gradually lost the power of standing, and for six months has been unable to do more than crawl about. In addition to the pains in the legs, he had, a year ago, partial loss of sensibility in both thighs; but after two months this was fully restored. No bladder or rectal troubles have occurred. His speech has not been so distinct for the last two years, according to his brother.

Family History.—He is the eldest in a family of six. James, John and Margaret, aged respectively 21, 20 and 19 years, are all healthy. Arthur, 17 years old, is the one who has the same disease, and his case is described later. Mary, the youngest, is 15 years old, and may be another case, for her brother writes that at times she is not steady on her legs. She did not, however, come under our observation. The father, a sergeant, had served nine years in India, but had no disease; he died from acute pneumonia at the age of 54. The mother, 54, is healthy. Grandparents shewed no nervous disease; on the father's side, grandfather is alive, aged 86; grandmother died at 47 of a "weakened heart." On the mother's side, grandfather died at 54, of "chills and dropsy"; grandmother died at 72. In no branch of the family is there any history of nervous disease.

Examination.—Patient has a peculiar physiognomy (figs. 1 and 2). The face is long and narrow, the figure deformed.

Deformities.—There is advanced spinal curvature, of the kypho-scoliotic variety, with the convexity to the right in the dorsal region (fig. 3). The feet show the peculiar deformity characteristic of Friedreich's ataxia (fig. 4). The plantar arch is greatly exaggerated, so that the condition resembles talipes equinus; but there is no contraction of the tendo Achilles or drawing up of the heel; it is a drooping of the front part of the foot, that is, an excessive flexion at the tarso-metatarsal joint. The arching is partly reduced by the weight of the body in standing, but mostly permanent, and due to contraction of the plantar ligaments; he wears ordinary boots, left unlaced over the instep, and his toes enter but a little way into the toe of the boot. The great toes are strung back hyper-extended; there is also a slight varus tendency in the feet; all these changes are more marked in the left foot. The hands present a slight degree

FIG. 1.

FIG. 2.

FIG. 3.

FIG. 4.

of the "main en griffe," the ring and little fingers being somewhat clawed (fig. 2). The metacarpus also appears too broad for the fingers.

Coordination. He is quite unable to assume or maintain the erect posture; he can get out of bed, cling to the side, and, reaching hold of neighbouring objects, guide himself about the room; he lifts his feet as if they were sticking to the ground at each step, and hobbles along. On attempting to stand alone, swaying movement of the body occurs, which would end in his falling backwards, if unsupported; this does not seem more marked when his eyes are shut; while sitting up in bed he is not steady. He is unable to thread a needle or perform other accurate movements of the hands, showing that ataxia is also present in the upper limbs.

Voluntary power appears to be normal in the arms, but is decidedly reduced in the legs, especially in flexion at the hip joint. The limbs are thin, but the muscles nowhere show any localised atrophy.

Reflexes.—Plantar reflexes are greatly exaggerated; knee-jerks could not at first be got when he lay in bed; but when sitting on the edge of the bed, with someone behind to lean against, they could be elicited on both sides; they were found to be somewhat of the spastic type, the whole of the limb going into a short spasm on tapping the tendon, and were more active on the left side.

Ankle clonus was obtained on the left side; three or four distinct contractions could always be produced. This point was confirmed by Dr. Kerr, and was constant whenever tested for; it is of great interest, as being a very unusual symptom, as also is retention of the knee-jerks, in Friedreich's ataxia.

Vaso-motor disturbance was evinced by the coldness of the extremities and the lividity of the hands.

Slight *nystagmus* occurred on wide lateral excursion of the eyes.

Speech was found to be characteristically affected, having a hesitating and peculiar careful plodding character, hastened at times, at the sacrifice of the end of a word.

Intelligence was above the average, and mentally he was very collected.

The following conditions were ascertained to be normal, viz.:—The pupils in size and reaction; the fundi; the perimetric tracings of the fields of vision; special senses; sensibility to touch, pain, and temperature everywhere; muscular sense; electrical reactions

of muscles, the bladder and rectal functions. The other systems of the body were healthy.

Progress.—Under observation he improved a little, so that he could stand against the bed and take his coat off, and he could thread a needle. Plantar reflexes became normal instead of exaggerated. Pain was experienced several times in the shin of the right leg. Symptoms of thyroidism came on after nine days' treatment with tabloids; nux vomica was then given, and for the last three weeks he went through Roth's spinal exercises.

After leaving the Infirmary he remained in the same condition till November, 1898, when he says he had a severe attack of influenza, and has been quite helpless, having to be carried about and suffering great pain in the back and between the shoulders, that when he tries to stand he feels as if a very heavy weight were pressing on his feet. His handwriting is irregular, but has not changed much. His general health is fairly good.

(2) The second case is that of the youngest brother, Arthur R., aged 17, who came first under observation. He had come on account of a girdle sensation, following a wrestle with another lad ten days before.

History.—He had not been well for some years before this, for, when fourteen years old and growing fast, his back began to get curved, and has grown more so ever since. Two years later unsteadiness in the legs was first apparent, and has since then progressed; walking getting more and more uncertain. He has had no bladder or rectal troubles. The girdle sensation came on directly after a wrench to his back, from being thrown to the ground. It is situated about the ninth dorsal root zone, and goes away when he lies down.

On examination patient is not so tall as his brother, whom he does not resemble in peculiarity of features (fig. 1).

Deformity.—He has spinal curvature of the same kypho-scoliotic variety, but much less advanced; the convexity in his case is to the left in the dorsal region. The feet are long and narrow, but not deformed. In the right hand the little finger is somewhat clawed, and in both the metacarpus seems unduly broad.

Coordination.—There is an air of general unsteadiness about him, especially when agitated from self-consciousness; choreiform movements are also manifested, such as shrugging the shoulders, knitting and raising the eyebrows, and moving the scalp in an uneasy manner.

He has difficulty in maintaining the erect posture, but can

balance himself after swaying to and fro, and apparently as well with his eyes shut as open.

In walking he lurches along, tacking from side to side to steady himself by catching hold of the furniture. The trunk is fairly steady when he sits up in bed. The head, however, is never still, owing to ataxia of the muscles of the neck; it appears to wander from its natural position, and has to be jerked back at times to be righted; there is marked hypertrophy of the sterno-cleido-mastoid muscles. The tongue cannot be held out steadily for any time. He is unable to thread a needle, the ataxia affecting also the arms.

Voluntary power is normal in the arms, but reduced in the legs; the limbs are spare, but the muscles firm, and show no localised atrophy.

Reflexes.—Plantar reflexes are extraordinarily active; a mere touch caused the leg to be thrown up off the bed and tickles him very much; especially active on the left side. Knee-jerks quite absent; ankle clonus absent; abdominal and epigastric reflexes brisk.

Slight *nystagmus* was produced on wide lateral movement of the eyes.

Speech is affected somewhat, being uncertain and wavering, though less laboured than his brother's.

Intelligence is above the average, and patient is of a nervous temperament.

Pupils were usually dilated, but reacted normally to light and accommodation. No hyperæsthetic zone corresponding to the girdle feeling could be found; nor any rigidity or tenderness of the spine.

The following points were found to be normal, viz.:—The fundi; perimetric tracing of fields of vision; special senses; sensibility to touch, pain, and temperature everywhere; muscular sense in judging weight; electrical reaction of muscles and the bladder and rectal functions.

The other systems of the body were unaffected.

Progress.—Under observation there was no great change, except that he learned to walk a little more steadily on the polished floors and to thread a needle. The state of the reflexes remained unchanged after full doses of nux vomica; latterly he performed Roth's exercises for spinal curvature. Nine months after he left he is reported to be about the same, except that his back is rather straighter. He occasionally experiences the girdle sensation.

Remarks.

We thus see that, in the elder brother, the deformities of the back and feet, together with the ataxia, affection of speech, and nystagmus, presented a most characteristic picture, but the peculiarity was in the increase of myotatic irritability in the legs. While the younger had choreiform movements, ataxia, loss of knee-jerks, &c., but the skeletal deformities less marked. In both cases the left leg was more affected than the right. There were no ocular symptoms. The slight disturbances of sensation, viz., pains in the legs, transient anæsthesia in the thighs, and girdle sensation, may be noted, and, lastly, the fact that the ataxia did not appear until two years after the spinal curvature in both cases.

In the *Dublin Journal of Medical Science*, May, 1895, Mr. J. M. Nolan records "Three cases of Friedreich's Disease, associated with genetous Idiocy." In one of these there was ankle clonus and exaggerated knee-jerks; in the other two knee-jerks and ankle clonus were absent. I have seen a case similar to these myself, in a child six years old, mentally deficient from birth, who had marked ataxia, being hardly able to stand and had loss of knee-jerks; the pupil reactions were normal. A brother of this child had died a few years before, and was reported to have been idiotic and never able to walk.

Such cases as these may be allied to Friedreich's disease, but that term, I hold, belongs as yet to a definite body of symptoms and signs, as depicted by Friedreich, and found since to be strikingly constant.

But in these cases some of these classical features were absent, uncharacteristic symptoms, such as enlargement of the thyroid, were present, the mental condition was the reverse of what usually occurs, and the disease was congenital instead of appearing first in childhood or adolescence.

Still, they show, as do those I have detailed, that occasionally the lesion may be so distributed, as, in one case, to produce increase, and, in another, destruction of myotatic irritability among members of one family otherwise similarly affected.

ON CERTAIN RELATIONS OF THE CEREBRUM AND CEREBELLUM (ON RIGIDITY OF HEMIPLEGIA AND ON PARALYSIS AGITANS).

BY J. HUGHLINGS JACKSON, M.D., F.R.S.

As the subject of the relations of the cerebellum and the cerebrum to the spinal motor centres, and by them to the muscles, is—in consequence of Dr. Charlton Bastian's important researches on the effects of total transverse lesions of the supra-lumbar cord—now receiving considerable attention, I have asked permission of the Editor of BRAIN to republish in its pages two articles I wrote a little more than twenty years (1877 and 1878) ago, one on "Rigidity in Hemiplegia," and one on "Paralysis Agitans" (Parkinson's Disease).

REMARKS ON RIGIDITY[1] OF HEMIPLEGIA.

(*The Medical Examiner*, April 5, 1877.)

With reference to several cases of hemiplegia, Dr. Hughlings Jackson expressed the opinion that the rigidity in hemiplegia is not owing to the cerebral lesion nor to the lateral sclerosis. There is evidently a duplex symptomatic condition, negative and positive; with loss of power over the muscles there is tonic action of them. Whilst the primary cerebral lesion can account for the paralytic element—the negative condition—it cannot (nor can the sclerosis in the lateral column) account for the tonic condition of the muscles, the positive element. Negative states of the nervous centres cannot *cause* positive states of muscles, they may *permit* them. His speculation is that the rigidity is owing to unantagonised influence of the cerebellum. He believes that in health the

[1] 1899. In some cases of hemiplegia there is a kind of pseudo-rigidity; I mean a rigidity from joint disease ("locking of the joints"), with no or very little muscular rigidity. I think this pseudo-rigidity occurs far oftener with left than with right hemiplegia.

whole of the muscles of the body are doubly innervated—innervated both by the cerebrum and cerebellum; there being a co-operation of antagonism betwixt the two great centres. Whilst the cerebrum innervates the muscles in the order of their action from the most voluntary movements (limbs) to the most automatic (trunk), the cerebellum innervates them in the opposite order. This also is supposed to be the order of their degree of influence on parts; and is equivalent to saying that the cerebellum is the centre for continuous movements and the cerebrum for changing movements. Borrowing medical terms to describe normal physiological conditions, the former are tonic the latter clonic. The muscles which on the above suggestions, are chiefly innervated by the cerebellum, are those which have the fewest different, the most tonic, the most "confluent" movements; the reverse for those which the cerebrum chiefly innervates. Thus, in walking, the cerebellum preserves the equilibrium of the body, tends to "stiffen" all the muscles; the changing movements of walking are the result of cerebral discharges overcoming in a particular and orderly way the otherwise continuous cerebellar influence. This is what is meant by a "co-operation of antagonism." The cerebrum does most "work," the cerebellum does little (if any) "work"; from continuous action of muscles balancing one another there is dissipation of energy (heat); from cerebral action there are molar movements (work).

When, however, the influence of the cerebrum is permanently taken off by disease of the cerebrum, as in hemiplegia, from the parts which it most specially governs [arm and leg], the cerebellar influence is no longer antagonised; there is unimpeded cerebellar influx and hence rigidity of the muscles which in health the cerebrum chiefly innervates. The principle, he thinks, accounts for the duplex, negative and positive, conditions in the contractures of hemiplegic children, in the athetosis of Hammond, and accounts for the rigidity and "propulsive" walk in paralysis agitans; temporary removal of cerebral influx also for tetany and certain hysterical attacks, and also for some post-epileptic conditions of movements. Physiological experiments were adduced in favour of the doctrine that two impulses arriving at a centre interfere with one another; the opposite effects of ligature in epileptiform seizures and in tetany were spoken of.

The above hypothesis is, Dr. Hughlings Jackson considers, only an application on a large scale of Thompson Dickson's[1]

[1] 1899. The "Principle of Loss of Control" was, I believe, first stated by Bernard and afterwards by Anstie, and then by Thompson Dickson.

Principle of Loss of Control, a principle applicable to all nervous centres, as is, Dr. Jackson thinks, the physiological Principle of Antagonism. In healthy operations the two principles are displayed; did not centres "resist" one another there could be no independence of operations; differentiation would be practically anulled. On the other hand, if some centres could not overcome the resistance of others—discharge them by their discharges—there would be no association of operations, differentiation would be useless; and if there were not some order of "resistances" and of "dischargeabilities" there would be no orderly co-operation.

On Cerebral Paresis or Paralysis, with Cerebellar Tremor or Rigidity.

(*The Medical Examiner*, March 28, 1898.)

In the *Examiner*, April 5, 1877, are reported some remarks by me on the interpretation of the rigidity in hemiplegia. I then suggested that the condition is duplex, that there is a negative condition, "cerebral paralysis," due to the destructive lesion in the corpus striatum, and a positive condition "cerebellar rigidity," due to increased cerebellar influx. The hypothesis starts with the assumption that the spinal centres receive impulses from both the cerebrum and cerebellum, which impulses in health interfere with one another (inhibit one another). If so, we should not say that the cerebral lesion, nor the wasting in certain spinal tracts consecutive upon it, *causes* rigidity; negative lesions can only cause negative conditions in muscles; in this case the paralysis. But on the hypothesis the cerebral lesion, in other words, loss of cerebral influence on the spinal centres, may *permit* the rigidity, for then the cerebellar influence is no longer interfered with, and, metaphorically speaking, "flows into the parts deserted by the cerebral influence." Hence it is better to say "unantagonised cerebellar influx" than "increased cerebellar influx." The cerebellum is healthy, or if over-active in effect,[1] it

[1] 1899. I have been supposed to hold that there is disease of the cerebellum in cases of paralysis agitans. On the contrary, as will be seen later in the text, my hypothesis implies that in this malady there is integrity of the cerebellum; the tremor and rigidity of paralysis agitans are, I suppose, the outcome of activities of perfectly healthy structures, cerebellum and anterior horns.

I do not believe that any *continuous* and *positive persisting* symptoms—positive symptoms so continuous and *persistent* as the tremor and rigidity of paralysis agitans—are ever the *direct* result of disease—of disease in the proper sense of pathological process. I think that, like those of paralysis agitans, they are always the *indirect* result of disease, and that they are directly the result of activity or of over-activity of healthy structures, that they are problems in the domain of physiology, not of pathology.

is not so because its cells have become morbidly over-unstable, but because there is less or no resistance to overcome, or, as we say, less or no cerebral influence to antagonise.

Mutatis mutandis, the same explanation applies to rigidity of the spinal and other muscles in cases of destroying lesions of the middle lobe of the cerebellum; there is then "cerebellar paralysis" and "cerebral rigidity."

I wish now to show how the hypothesis applies to the interpretation of several symptoms of paralysis agitans.

The regional distribution of this motor affection points unmistakably to the cerebrum (whether of the motor tract or cerebrum proper is not said) as the seat of the morbid anatomy. It is essentially hemiplegic, although very commonly it *becomes* two-sided. I believe that we have here, too, a minus cerebral and a plus cerebellar condition. It is sometimes asserted that there is no paralysis in what is called *paralysis* agitans; but some of those who say so add that the movements are slow, and slowness of grasp is as much evidence of paralysis as weak grasp is. To ignore the element of rapidity in estimating the "momentum" of a mobile part would be like ignoring the velocity of a cannon-ball in estimating its momentum. It is certain that we have tremor and rigidity.[1]

On the supposition that the cerebral influence is diminished, we may explain the tremor—negative clonic cerebral spasm (most of the spasm in ordinary epileptiform seizures being positive clonic cerebral spasm). On the supposition that the cerebral influence is taken away altogether, we may explain the rigidity. Tremor, at least rhythmical tremor, is a minor degree of tonic spasm. If, experimentally, shocks at comparatively long intervals are sent into a muscle, there is a sequence of contractions, the analogue of tremor; but if they be sent in at exceedingly short intervals, there is tetanic contraction, the analogue of rigidity. Thus the tremor of paralysis agitans differs from the rigidity of hemiplegia, not fundamentally, but in degree; in the former the cerebral influx is diminished; in the latter it is taken off altogether. And as the disease progresses, or, as the hypothesis has it, when the cerebral influx ceases entirely, then rigidity of the parts *does* ensue, which in tremor were approaching rigidity.

(1) In health the cerebellar influx is fully antagonised; in (2) the early stages of paralysis agitans it is intermittently antagonised—the movement constituting each single tremor occurring

[1] In a few cases there is no tremor, or scarcely any; there is rigidity only

betwixt the cerebral impulses; in (3) late stages it is not antagonised at all, and there is such a stream of cerebellar impulses that rigidity occurs. We have cerebral paresis with cerebellar tremor; later, cerebral paralysis with cerebellar rigidity.

So much for the parts which the cerebrum *chiefly* innervates in health, and which it slowly "abandons," and which, in consequence, the cerebellar influx gradually "invades."

In paralysis agitans there is "loss of expression" of the face, but this so-called "loss" of expression of the face is one *fixed* (starched) expression. An unvarying expression, however caused, is in effect a loss of expression. The hypothesis is that there is some facial paralysis "caused" by the cerebral disease, and some facial rigidity, owing to "permitted cerebellar influx."

On the hypothesis (see *Examiner*, April 5, 1877) the spinal muscles are those which the cerebrum influences least and the cerebellum most; thus there is rigidity without prior tremor. This is manifested by the well-known attitude, and indirectly by the propulsive gait. The rigid state called "attitude" of the body, face, and limbs is more characteristic of the disease than the tremor, in spite of the name "paralysis agitans."

This is a mere outline.[1] It may be remarked that the general attitude often seen in cases of paralysis agitans (face, arms, and legs) is like that seen in cases of rapid locomotion; it is at least a good caricature of the position, supposing locomotion to be suddenly arrested, and parts of the body remaining fixed. In running, the body is bent forward, the arms are flexed, &c., and the facial muscles come into vigorous action.[2]

As before said (*op. cit.*) the hypothesis is applied to cases of athetosis, and to the mixed condition of paralysis with twisting movements in persons hemiplegic from early life.

For *all* morbid over-mobile conditions of one side I have suggested the name hemikinesis, although I have been supposed to use it only for the athetosis of Hammond. But we have two different sub-classes; the hemikinesis due to cerebral discharges (unilateral chorea and unilateral epileptiform seizures), and the hemikinesis owing negatively to cerebral destroying lesions with (positively) permitted cerebellar influx.

[1] With the assistance of my clinical clerk, Mr. Whitford, I tried, a few days ago, the effects of ligature on a patient, the subject of paralysis agitans, the tremor being on both sides. At first trials we obtained arrest of the tremor of the arms, even when the ligature was applied round one calf; but at the second *séance* we got no such results. The plan is worthy of trial in slighter cases.

[2] 1899. The attitude in an advanced case of paralysis agitans is something like that of a feeble old man when he is going upstairs.

Remarks, 1899.

I have written on this subject several times since 1878. (*Med. Times and Gazette*, February 12, 1881; Brain, January, 1888, p. 312 *et seq.*; *Brit. Med. Journal*, March 5, 1892; *Lancet*, December 12, 1896, and January 8, 1898.) In these papers I have acknowledged all the difficulties I knew of.

To the hypothesis propounded in the two papers reproduced from the *Medical Examiner*, it was objected that the "fact" that upon complete transverse lesion of the cord above the lumbar enlargement (whereby both cerebral and cerebellar influences are cut off from motor centres below that transverse lesion) the knee-jerks are exalted and that the legs are, or soon become, rigid—that these super-positive conditions exist when the cerebellum is altogether excluded—disprove the hypothesis.

I was so impressed by this "fact," as I then took it to be, that[1] I went a long way towards abandoning the doctrine of cerebellar influx, if not all the way. I then wrote: "I admit . . . that since after section of the cord right across, destroying communication with both cerebrum and cerebellum, there is increased reflex activity of the parts below, we cannot suppose that unantagonised cerebellar influx is essential for the conditions mentioned. I make this admission in consequence of some verbal criticisms by Ferrier."

Since I wrote that, Dr. Charlton Bastian (Quain's "Dictionary of Medicine," p. 1480 (1882), "Paralyses, Cerebral, Bulbar and Spinal," 1886,[2] and also "On the Symptomatology of Total Transverse Lesions of the Cord," *Med. Chir. Trans.*, 1890), has done far more important work on the matter than I have. He has shown that upon a complete transverse lesion of the cord above the lumbar enlargement there is, contrary to what, heretofore, had been universal physiological

[1] "On a case of Temporary Left Hemiplegia with Foot Clonus and Exaggerated Knee Phenomenon, after an Epileptiform Seizure beginning in the Left Foot," *Medical Times and Gazette*, Feb. 12, 1881.

[2] In this work, "Paralyses, &c.," 1886, p. 538, Bastian writes, ". . . with *a total transverse* lesion in the lower cervical region, nearly all reflexes dependent upon lower portions of the cord are *abolished rather than exaggerated*."

and medical doctrine, flaccidity of the legs and loss of the knee-jerks.

Bowlby (*Med. Chir. Trans.*, 1890), Thorburn (*Med. Chronicle*, May, 1890), and Bruns, of Hanover (*Archiv für Psychiatrie*, Band xxv., pp. 1-72), have confirmed Bastian's conclusions.[1] I do not imply that the writers just mentioned accept my hypothesis of the nature of the dynamical relations of the cerebrum and cerebellum. My hypothesis is rejected by Bruns, also by Gerhardt, and by Ferrier and other eminent neurologists.

Dr. Charlton Bastian's conclusions as to the state of the muscles and tendon reactions of parts below a *complete* supra-lumbar transverse lesion of the cord, are now admitted by nearly all medical neurologists to be correct.[2] The real "facts" of cases of the complete transverse lesions mentioned are not against the hypothesis I put forward in 1877-8. Bastian wrote (*Med. Chir. Trans.*, vol. lxxiii., 1890, p. 208) :—"I came to the conclusion that a notion closely akin to that of Dr. Hughlings Jackson was most capable of explaining all the facts." After considering the then (1890) current views as to the causation of rigidity and exaggerated tendon reactions in hemiplegia, Bastian wrote (p. 209) :—"Great difficulties formerly stood in the way of explaining many of the facts without the aid of some such views (although grave objections could always be alleged against them); hence the few adherents which the counter-explanation of Dr. Hughlings Jackson has hitherto been able to command. Now, however, it seems to me that the new facts established in this paper will be found to be

[1] See a most able paper, "On the Conditions of the Reflexes in Total Transverse Division of the Spinal Cord," by Ernest S. Reynolds, BRAIN, vol. xviii., p. 148 *et seq.*, 1895.

[2] Sherrington writes in a very remarkable and most valuable paper (*Trans. Royal Society*, vol. cxc., B., 1898, pp. 144-145) :—"My results on Monkeys bear out strikingly and fully what Bastian describes as the typical condition in Man after complete transverse destruction across the cord. The chief difference is that in the Monkey in most cases—partly, perhaps, because the lesion is more localised by experimental infliction than by accidental—the depression is not so severe. For instance, the knee-jerk, which disappears almost immediately after the transection, returns usually in a week or ten days (Sherrington, *Journ. of Physiol.*, 13, 1892; A. S. F. Grünbaum, *Journ. of Physiol.*, 16, p. 368); often, however, not for three weeks; occasionally, on the other hand, in ten minutes."

altogether opposed to the fashionable views above cited, and to be just as much in favour of some modification of the doctrine of Hughlings Jackson."

In the second reprint (*Medical Examiner*, 1878) I spoke of localisation of the disease in paralysis agitans, suggesting that the regional distribution of this motor affection points to the cerebrum. I have, since 1878, submitted the hypothesis that in this malady there is slowly-spreading atrophy of cells of the Rolandic region of the cortex in the order of their size, from smallest towards largest. Presumably these cells, in their order from smallest to largest, represent muscles,[1] in their order from smallest towards largest. In paralysis agitans the interossei[2] are the muscles of the hand which are preponderatingly affected, and the set of the hand resembles that of the tetany of children. In most cases of ordinary hemiplegia with rigidity there is destruction of fibres of some part of the course of the pyramidal tract (internal capsule), a comparatively random and brutal lesion (clot or local softening). In most cases of this hemiplegia with rigidity the set of the hand is not interosseal, but is due especially to contraction of the large flexors and extensors; this set of the hand a little resembles that of the hand in carpo-pedal contractions of children. I believe that both tetany and carpo-pedal contractions are owing to loss of cerebral control permitting greater cerebellar influx, and surmise that, in children, the differences in the disposition of the thumbs and fingers and of the hands in carpo-pedal contractions and tetany, depend, at least partly, on differences in the degree of development (or of perfection) of the pyramidal tract and cortex; the nervous arrangements for the intrinsic muscles will, I imagine, be later in their development or perfection than those for the large flexors and extensors of the hand.

A case, very important in many ways, has been published by Strümpel (*Deutsche Zeitschr. f. Nervenheilk.*, B. v., 1894) of a woman whose attitude was very like that of a subject of advanced paralysis agitans. Brissaud (*Leçons sur les Maladies Nerveuses*,

[1] Properly "small and large movements." See my second Lumleian Lecture, *Brit. Med. Journ.*, vol. i., 1890. For important remarks on the significance of sizes of nerve-cells see Bevan Lewis "On Mental Diseases," 2nd ed., 1899.

[2] In some cases of paralysis agitans there is over-contraction of the interossei, and thus hyper-flexion of the first phalanges and hyper-extension of the other phalanges of the fingers. See a very interesting paper, *Des Déformations de la Main*, by J. E. Siotis. I need scarcely remind the reader of Charcot's important researches on paralysis agitans, in which, among many other things, he considers the set of the hands in cases of paralysis agitans, likening the disposition of the fingers in some cases to that in some cases of chronic arthritis.

Salpêtrière, 1893-4) reproduces (p. 45 *op. cit.*) a portrait of this patient, and (p. 486 *op. cit.*) says of it:—"Est-il possible d'imaginer une attitude et une physionomie plus *parkinsoniennes ?* C'était une pseudo-bulbaire, puisqu'elle avait le syndrome d'une paralysie labio-glosso-laryngée spasmodique, causée par une double lesion systematique du faisceau paramidal." (See also Brissaud, *op. cit.*, pp. 41 *et seq.*) There was no tremor; there was rigidity of face, tongue, neck, arms and legs. There was difficulty in turning the eyes to the left, and ultimately all ocular movements were lost. Strümpell writes (*op. cit.*):—"On being asked, the patient is unable to carry out the smallest movement in any part of the body or with the eyes. But of her own accord she occasionally makes a slight movement with the eyes or with the eyelids." No morbid changes were found in the cells of the cortex cerebri. Degeneration was traced upwards beyond the upper end of the internal capsule, but could not be followed to the cortex cerebri. The lesion in Strümpel's patient was a slowly-developed one, whereby cerebral control would be slowly taken off anterior horns, and consequently, as I suppose, there would be permitted gradually greater cerebellar influx on those parts (anterior horns) from which the cerebrum was being, by the lateral sclerosis, slowly cut off. I do not say that this case was one of paralysis agitans, but I think it illustrates, if it does not confirm, the *principle* of the hypothesis I have put forward as to the pathology and physiology of that malady.

In cases of paralysis agitans, as in so many other nervous maladies, we have to bear well in mind Broadbent's hypothesis,[1] 1866. Bearing it in mind, I should say that double hemiplegia is more than the double of hemiplegia. In hemiplegia the bilaterally-acting muscles of both sides escape, or suffer very slightly, whereas in double hemiplegia the bilaterally-acting muscles of both sides suffer as well as do the unilaterally-acting muscles of both sides. Hence, in Strümpel's patient (symptomatically a case of double hemiplegia with rigidity), there was, as Brissaud puts it, pseudo-bulbar paralysis; there was involvement of very important bilaterally-acting muscles of both sides.

[1] I have profited very greatly by the application of Broadbent's hypothesis to the explanation of complex neurological problems. Thirty years ago (*Med. Times and Gazette*, Aug. 15, 1868) I wrote, under the heading "Remarks on Broadbent's Hypothesis," to urge those who observe defective bilateral movements with hemiplegia—particularly difficulty in swallowing—to enquire carefully if the patient has not been previously paralysed on the other side. Again, at an autopsy on a patient known to have been paralysed of *but one side*, and who had had difficulty in swallowing, we should search both sides of the brain for evidences of disease.

An advanced case of paralysis agitans *is* double hemiplegia with rigidity (and some tremor, which is an approach to rigidity). It will be found in some cases of this malady that, if there be not bulbar (pseudo) paralysis, there is involvement of other bilaterally-acting or alternately-acting muscles—rigidity of those of the face, neck, back, and abdominal wall. The hypothesis may be considered from another standpoint.

In an early stage of etherisation for surgical purposes there is muscular rigidity, and the knee-jerks are exalted. I think it possible that this occurs when the small cells of the cerebral cortex have succumbed to the ether, and when the large cells of the cerebellum have not been rendered functionless by it. In this stage, according to my hypothesis, there is negatively loss of some degree of cerebral control, and positively there is permission of a greater degree of cerebellar influx. In later stages, when I suppose the large cerebellar cells also have been rendered functionless, there is flaccidity of muscles and loss of the knee-jerks.

The situation, however, is complicated. The ether has, so to say, no malice against any particular part of the nervous system, but will put *hors de combat* small nerve-cells, wherever they are (cerebral, spinal, or cerebellar), sooner than it will large cells, wherever they are.

Dr. Hewitt, in his remarkably interesting work, "Anæsthetics and their Mode of Administration," p. 154, says of etherisation: —"In exceptional cases a fine tremor, known as 'ether tremor,' may occur." Perhaps the annulling of function of cells of the cortex is not solely by ether or chloroform, but both by the anæsthetic used and by supervenous blood (deficiency of oxygen). I say this in consequence of what Dr. Hewitt writes, p. 281 *op. cit.*:—"Rigidity will often disappear when more air is allowed with the anæsthetic, a fact which leads one to suppose that the condition may sometimes depend upon intercurrent asphyxiation." If so, there may be removal of cerebral control, partly by the action of the anæsthetic and partly by that of supervenous blood. But there is a difficulty to my hypothesis, so far as supervenosity is concerned. Risien Russell has found that the knee-jerks in dogs are exaggerated in early stages of artificial asphyxia, even when the supra-lumbar cord has been divided. It may be that in rigidity under anæsthetics, &c., super-venous blood, which annuls the function of cortical cells (of the Rolandic region at least) and which stimulates some medulla centres (respiratory, &c.), may stimulate some of the cells of anterior horns, and thus produce the rigidity or part of it.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF LONDON.

At a Pathological Meeting held on Thursday, December 7, 1899, at 11, Chandos Street, W., the following communications were made :—

(I.) Dr. F. E. Batten and Dr. J. S. Collier.—*Spinal cord changes in cases of cerebral tumour.*

The object of the paper was to show that degeneration of the posterior columns of the cord frequently occurs in cases of cerebral tumour. The authors have examined twenty-nine cases of cerebral tumour occurring in patients of all ages and in varying situations, and they find that degeneration of the posterior columns to a greater or lesser degree occurs in about 65 per cent. of the cases. A short abstract from the papers of previous observers was then given, and the suggested explanation of the changes found discussed. Of these, the most important were (1) a toxic process; (2) increased intra-cranial pressure; (3) venous stasis, anæmia, or thrombosis; (4) retrograde degeneration.

The character of the degeneration was such that though readily shown by the Marchi method it was not obvious by the Weigert-Pal method. The degeneration affects the posterior columns of the cord, and more especially the postero-external columns in the lower cervical region, but in certain cases the degeneration affects the lumbar and even the sacral region of the cord. The degeneration can be traced up to the posterior column nuclei. The degeneration is of root origin, as shown by certain cases where only a few roots are affected; the degeneration is limited to this root area, and also by the fact that the endogenous tracts of the cord tend to escape, although it cannot be said that they are perfectly free from degeneration. (Few will deny, however,

that these endogenous tracts also contain fibres of exogenous origin.)

The posterior nerve roots usually show some degeneration, but always considerably less than the degeneration in the cord, and they may in some cases show none.

The posterior root ganglia appear perfectly normal.

The degeneration neither depends on the situation of the tumour nor on the nature of the growth, but it is dependent on the increase of intra-cranial pressure. An increase of the intra-cranial pressure tends to distend the sub-arachnoid space in the spinal cord, and such distension puts traction on the posterior nerve roots. It has been shown by other observers that the posterior nerve root is especially thin and constricted at the point where it enters the spinal cord. Traction would therefore tend to injure the roots especially at this point. It is therefore argued that the degeneration in the cord is an ascending degeneration, while that in the posterior roots is a retrograde change. Such a view is borne out by the present series of cases, since, while there is marked degeneration in the posterior columns of the cord, littl or no degeneration can be seen in the posterior roots.

Certain clinical features were next dealt with, viz., the effect of operation, the condition of the knee and arm-jerks, and the presence of optic neuritis.

The following conclusions were arrived at :—(1) That degeneration of the posterior columns of the spinal cord frequently occurs in cases of cerebral tumour, *i.e.*, about 65 per cent.; (2) that such degeneration is more liable to affect the spinal cord in the cervical region than in the dorsal or lumbar, and is more marked in the postero-external than in the postero-internal columns; (3) that such degeneration is of root origin, and arises from the point where the root enters the spinal cord, the posterior roots being always less affected than the posterior columns and may show no degeneration; (4) that such degeneration is due to traction on the posterior roots by the distension of the spinal arachnoid owing to the increased intra-cranial pressure; (5) that such degeneration is independent both of the situation and of the nature of the tumour except in so far as their liability to give rise to the preceding condition is concerned; (6) that optic neuritis bears no relation to the occurrence of posterior degeneration; (7) that the absence of the knee-jerk (except in the semi-comatose state) indicates posterior degeneration, but that their presence does not negative such a condition; and the absence of the arm-jerks probably has the same significance.

(II.) Dr. WILFRID HARRIS.—*An experimental investigation on the decussation of the optic nerves in various animals, and on the path of the reflex for the contraction of the pupil to light (Meynert's fibres).*

This work was undertaken as a necessary preliminary to the study of reflex iridoplegia in man, or the Argyll-Robertson pupil. Clinical study of the consensual reflex contraction of the pupil in cases showing uni-ocular Argyll-Robertson pupil prove that the usually accepted path for the light fibres from the corpus quadrigeminum to the third nucleus of the same side (the two nuclei being connected together), will not explain the results obtained; inasmuch if light be directed obliquely upon the temporal half only of the retina of the affected eye, then the impulses travel up only by the optic tract of that side, owing to the non-decussation of the optic fibres from the outer halves of the retina in man. In such case the pupil of the affected eye does not contract to light, whereas there is brisk consensual contraction of the opposite pupil. According to the accepted scheme this could only result through the impulses passing through the sclerosed Meynert's fibres or affected third nucleus of the damaged side, which is entirely improbable.

Now in the lower animals, fishes, amphibia, birds, and lower mammals, the optic nerves decussate completely, as shown by sections of chiasma of toad, chameleon, pigeon, owl, and rabbit. Studies of the pupil reflex to light in the toad, frog, pigeon, parrot, owl and rabbit, show that only the pupil on which light is thrown contracts reflexly. There is no consensual reflex to light in these animals. Therefore, since there is total decussation at the chiasma there must exist in addition a posterior total decussation for the pupil-reflex fibres between the optic lobes and third nuclei. This then is probably the primitive arrangement in animals without binocular vision.

Partial binocular vision however is obtained in warm-blooded carnivorous animals, which require especially keen sight to follow up their prey, as seen in hawks and owls, and amongst mammals especially in the cat tribe. Yet the decussation remains complete in all birds, as proved by sections of chiasmata in owls, and by degeneration following enucleation and Marchi staining in pigeons; and in these only the pupil on which light is thrown will contract reflexly. Partial decussation is seen first in mammals. Practically complete in rabbit, with no binocular vision and unilateral pupil reaction, only a very few fibres passing

up the optic tract of the same side; in the cat, with good binocular vision and consensual pupil reaction, degeneration results show apparently an equal number of degenerated fibres passing up the tracts of each side. Wounds of the optic tract and corpus quadrigeminum on one side in cats have produced hemianopia with hemianopic pupil reaction, and marked degeneration in Meynert's fibres, which decussate ventrally at level of third nuclei to form a tract ventral to the opposite posterior longitudinal bundle, with some degenerated uncrossed fibres in the posterior longitudinal bundle of the same side. These fibres are precisely similar to the degenerated fibres in Meynert's decussation in cats shown by Boyce after extirpating one hemisphere.

None of these fibres can be traced either into the third nuclei or third nerves. But in animals with semi-decussation at the chiasma and consensual pupil reflex the primitive posterior total decussation of the pupil-reflex fibres must either be modified so that the corpus quadrigeminum of one side is connected with the third nucleus of the same as well as the opposite side, or else the two third nuclei must be tied together so as to react consensually. And in birds it is evident that the third nuclei are not connected together as regards the light reflex, and in man the phenomena of uniocular Argyll-Robertson pupil already spoken of negative the possibility of any connection between the nuclei being the path for the consensual light reflex. Therefore the former arrangement, or posterior semi-decussation, analogous to the semi-decussation at the chiasma is theoretically the necessary path for the light reflex in cat and the higher mammals. Such a path is probably furnished by the semi-decussation of Meynert's fibres as shown by the degeneration experiments in cats; and the probable path of the light reflex is then as will be shown in a diagram.

(III.) Dr. MOTT and Mr. A. F. TREDGOLD.—*Hemiatrophy of the brain and its effects upon the cerebellum and spinal cord.*

Clinical.

J. E., male, aged 26, single; occupation, shoe-black. Considered to be a case of epilepsy with dementia. He has frequent fits, and has had them since childhood. He is very considerably demented, but subject to occasional sudden attacks of maniacal excitement. There is paralysis of the right arm and leg, with increased reflexes. Pupils are equal and react to light and accommodation. He died at the age of 30 of acute phthisis.

Post-mortem examination.

Skull.—The left side is much thicker than the right (in some places twice as thick), although there is no asymmetry when viewed from the outside.

Brain.—Marked wasting with enormous dilatation of ventricle of the left hemisphere, which weighs less than a third that of the right.

Cerebellum.—Atrophy of the right upper quadrant.

Spinal cord.—Notably asymmetrical, due to wasting of antero-lateral columns on left side.

Microscopical examination.

Brain (left hemisphere).—Great atrophy of basal ganglia, chiefly affecting the optic thalamus. Cortex as a whole considerably thinned; there is an absence of large pyramidal cells, and the polymorphous cells appear to be slightly deficient. The smaller pyramids do not seem to be affected. There is a very great diminution in the number of nerve fibres; most notably is this the case with those of the projective system. The commissural and association fibres are not nearly so much affected.

Cerebellum.—Here the atrophy is confined to the lobus culminis of the right upper hemisphere; the layer of large granule cells lying immediately underneath the cells of Purkinje are not diminished, and the interlacing (? association) fibres are not affected. There is an entire absence of the cells of Purkinje, and a very great diminution of the coarse bundles of fibres forming the rays. The right superior peduncle is but a quarter the size of the opposite side. In the *pons* and *medulla* there is great diminution in the fibres of the fillet and pyramidal tracts on the left side. The olives and restiform bodies are symmetrical. The posterior column nuclei and arciform fibres have not yet been examined.

Spinal cord is obviously asymmetrical to the naked eye, there being marked atrophy of the left side all the way down the cord as far as L. 1 segment, and the atrophy being especially pronounced in the antero-lateral columns.

The following is a brief summary of the changes found in the different tracts of the cord when examined microscopically.

Left direct pyramidal.—Some sclerosis extending down as far as the first dorsal segment. *Right crossed pyramidal* is sclerosed and contains many swollen fibres as far down as L. 4. *Antero-lateral* descending tracts on left side are markedly deficient as

compared with the opposite side. This condition can be seen as far as L. 3. There is some slight sclerosis at the periphery of the cord in the cervical region, but the chief condition in these columns is not one of sclerosis, but of deficiency—the tracts do not seem to be present at all. It is the absence of these tracts which gives rise to the marked asymmetrical appearance of the cord. The *direct cerebellar tracts* are normal. In the cervical region there is sclerosis in Goll's column, the left side being slightly more affected than the right. The spinal ganglia were examined by Nissl's method, but showed no change. *Burdach's columns* are healthy. *Cells of anterior horns.*—There is a marked diminution in the cells of the right side; indeed, it can readily be seen by the naked eye that the right anterior horn is atrophied. Sections stained by Nissl also show that many of the cells which are present are undergoing degenerative changes. It must be said, however, that this diminution of nerve cells appears to be confined to the cervical and lumbar regions. No notable difference can be detected between the two horns in the dorsal region. In the cervical and lumbar regions, whilst there is a general numerical diminution of cells on the right side as compared with the left, it does certainly appear that this diminution is most marked in those cells forming the lateral groups and those at the base of the horn, and this is especially so in the 8 C., 4 and 5 L., and 1 S. segments. In the affected horns the nerve fibres are also deficient.

Clarke's column.—In the 12 D. and 1 L. segments there are certainly fewer cells on the right side, but above this the two sides are quite symmetrical, and all the cells appear particularly healthy.

Rules

AND

LIST OF OFFICE BEARERS AND MEMBERS

OF THE

Neurological Society of London,

1899,

WITH THE

Annual Report of Council, and Balance Sheet for the Year 1898.

LONDON:
JOHN BALE, SONS & DANIELSSON, LTD.,
83-89, GREAT TITCHFIELD STREET, OXFORD STREET, W.
1899.

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Neurological Society of London.

REPORT OF COUNCIL.

FEBRUARY 2, 1899.

The Council has much pleasure in reporting the continued success and prosperity of the Society. The members now number 206.

The Inaugural Address was delivered at the Annual General Meeting on January 13, 1898, by Mr. VICTOR HORSLEY, F.R.S., on "The Degree of Discharge of Different Nerve Centres."

The vacancy caused on the list of Honorary Members by the death of Professor DU BOIS REYMOND, of Berlin, has been filled by the election of Professor HITZIG, of Halle, formerly one of the Corresponding Members of the Society, and the vacancy thus caused on the list of Corresponding Members has been filled by the election of Professor GUSTAF RETZIUS, of Stockholm.

A feature in the year's programme has been a visit, in April, to Cambridge, where, at the invitation of the Vice-Chancellor of the University, Dr. ALEX. HILL, formerly President of the Society, a Physiological Meeting was held. At the conclusion of the meeting the Society was most hospitably entertained by the Vice-Chancellor. This was the first occasion on which the Society has met out of London.

As during last year, the Proceedings have been published at greater length in "Brain" than was the custom in former years, and an abstract appended to this Report gives some idea of the value and character of the work of the Society during the past year.

The Council reports with regret the resignation of the Senior Secretary, Dr. FREDERICK MOTT, after four years of valuable service rendered to the Society.

Proceedings of the Society during the year 1898.

January 13th, Mr. VICTOR HORSLEY, F.R.S., Inaugural address, "The Degree of Discharge of Different Nerve Centres," *Brain*, vol. xxi., part lxxxiv.

February 24th, Dr. DONKIN (1) "Spastic Paraplegia in a Child aged 4 years;" (2) "Functional Hemiplegia with Hemianæsthesia in a Man aged about 50 years;" (3) "A Case of Paralysis Agitans." Dr. MOTT, "A Case resembling Scleroderma in which there was Diminished Sensibility over the Area supplied by the Two Inferior Divisions of the Fifth Cranial Nerve, with loss of taste over the anterior two-thirds of the tongue, and loss of smell." Dr. HERBERT HAWKINS, "Case of Peroneal Type of Muscular Atrophy." Dr. BEEVOR, "Case of Syphilitic Spinal Meningitis." Dr. LEONARD GUTHRIE, "Case of Unilateral Syringo-myelia with Bulbar Symptoms." Dr. STEPHEN MACKENZIE, "A Case of Nodding Spasms." Dr. ORMEROD, "A Case of General Athetosis." Dr. F. E. BATTEN, "Friedreich's Disease with Mental Defect." Dr. ALDREN TURNER, "A Case of Diplegia Facialis, with paralysis of both external recti, defect in finer movements of hands, spastic gait and double club-foot."

April 23rd, Dr. ALEX. HILL, "Some Specimens of Granules." Professor MACALISTER, "The Race-Characters of Australian Brains." Dr. LANGLEY, "Some Points relating to the Sympathetic System." Dr. GASKELL for Miss ALCOCK, "The Distribution of the Facial Nerve in Ammocœtes." Dr. F. E. BATTEN (1) "Nerve Terminations in Muscle Spindles;" (2) for Dr. SIHLER, of Ohio, "Motor Nerve Termination within the Muscle Spindle." Dr. MOTT, "Degeneration of the Neuron."

June 30th, Dr. C. E. BEEVOR opened a discussion on "The Accurate Localisation of Intra-cranial Tumours," and Drs. BYROM BRAMWELL, MICHELL CLARKE, and SHARKEY took part in the discussion.

October 27th, Drs. ORMEROD and F. E. BATTEN, "Idiopathic Muscular Atrophy (Landouzy-Déjérine type) in Mother and Son." Dr. BEEVOR, "Unilateral Wasting and Paralysis of the Lower Half of the Trapezius." Dr. SINIGAR, "Case of Idiopathic Muscular Atrophy." Dr. F. W. MOTT, "Arsenical Neuritis from Local Action of the Poison followed by general Degenerative Effects upon the Nervous System." Dr. ORMEROD, "A Case of Hereditary Chorea." Dr. LUNN and Mr. BALLANCE, "Case of Removal of Cerebellar Tumour, three years and eleven months after the operation" (case shown at a meeting of the Society eighteen months ago). Mr. LAKE and Mr. BALLANCE, "Case of Recovery from Temporo-Sphenoidal Abscess." Dr. BEEVOR and Mr. BALLANCE, "Case of Removal of Subcortical Cerebral Tumour." Dr. JAMES TAYLOR and Mr. BALLANCE, "Case of Trigeminal Neuralgia, in which removal of the Gasserian Ganglion was followed by complete relief."

December 15th, Dr. F. W. MOTT, "Changes in the Brain, Muscles, and other Organs, found in persons having died with prolonged epileptiform convulsions." Dr. F. E. BATTEN, "Congenital Abnormalities in the Nervous Systems of Three Children." Drs. H. H. TOOTH and RISIEN RUSSELL, "Acute Myelitis with Bacteriological Examination."

BALANCE SHEET, 1898.

Cr. NEUROLOGICAL SOCIETY. **Dr.**

RECEIPTS.	£	s.	d.
To Balance	46	5	6
Subscriptions for current year	192	4	0
Do. in arrears	12	0	0
Do. in advance	3	0	0
Share of profit on "Brain"	42	3	1
Contributions by Messrs. Macmillan towards cost of illustrations in "Brain"	15	0	0
	£310	12	7

EXPENDITURE.	£	s.	d.	£	s.	d.
By Stationery and Printing—						
Pulman & Sons	1	15	8			
Bale & Danielsson ...	20	5	9			
				22	1	5
Illustrations—Bale & Danielsson ...				89	7	6
Microscopes—Baker				1	1	0
Hire of Rooms and Refreshments—Medical Society				20	15	0
Messrs. Macmillan for publishing "Brain"...				110	16	6
Petty Cash				0	15	0
Balance				65	16	2
				£310	12	7

Audited and found correct, December 6, 1898,

CHARLES E. BEEVOR, *Hon. Treasurer.*

R. PERCY SMITH.
HENRY HEAD.

RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF LONDON.

2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Corresponding, and Honorary Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.

8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.

10.—The Council shall nominate candidates for election into the Society as ordinary Members from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Pound, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due, shall be considered as equivalent to resignation.

15.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, and ten Councillors.

16.—Five Members of the Council shall form a quorum.

17.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

18.—The office of Vice-President shall be tenable for two years; one Vice-President being elected every year.

19.—The Secretaries and Treasurer shall be elected annually with eligibility for re-election.

20.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

21.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

22.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

23.—There shall be six ordinary meetings annually, of which the first held in each year shall be the Annual General Meeting.

24.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

25.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty Members.

26.—The President shall have the power of inviting any person to attend and to take part in the scientific work of the meeting.

27.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a Member, they shall submit the question to a special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him, and he shall thereupon cease to be a Member of the Society.

28.—The Council shall draw up and submit for the approval of the Society, supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

29.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of "BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by A. de Watteville, M.A., M.D., B.Sc.

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„ Hitzig, Halle.
Sir Samuel Wilks, Bart., London.

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„ Grasset, Montpelier.
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„ Obersteiner, Vienna.
„ Retzius, Gustaf, Stockholm.
„ Ribot, Paris.
Dr. Weir Mitchell, Philadelphia.

LIST OF PRESIDENTS FROM THE FOUNDATION OF THE SOCIETY.

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1887 Sir Samuel Wilks, Bart., M.D., F.R.S.
1888 Sir James Crichton-Browne, M.D., F.R.S.
1889 Jonathan Hutchinson, F.R.S.
1890 Thomas Buzzard, M.D.
1891 John S. Bristowe, M.D., F.R.S.
1892 Henry C. Bastian, M.D., F.R.S.
1893 Edward A. Schäfer, F.R.S.
1894 David Ferrier, M.D., F.R.S.
1895 Sir William H. Broadbent, Bart., M.D., F.R.S.
1896 Alexander Hill, M.A., M.D.
1897 George H. Savage, M.D.
1898 Victor Horsley, M.B., F.R.C.S., F.R.S.
1899 W. H. Gaskell, M.D., F.R.S.

LIST OF HUGHLINGS JACKSON LECTURERS.

1897 J. Hughlings Jackson, M.D., F.R.S.

*** Members are requested to communicate with the Secretaries when corrections are necessary.

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EXPLANATION OF ABBREVIATIONS.

O.M., Original Member.	Tr., Treasurer.
Pres., President.	Sec., Secretary.
V.P., Vice-President.	C., Member of Council.

H.J.L., Hughlings Jackson Lecturer.

ORDINARY MEMBERS.

Elected.

1892 ALEXANDER, ROBERT REID, M.D., The Asylum, Hanwell, W.

1897 ANDERSON, H. KERR, M.B., B.Sc., 9, Park Terrace, Cambridge.

1894 ANDRIEZEN, W. LLOYD, M.D.

1891 BALLANCE, C. A., M.S., F.R.C.S., 106, Harley Street, W. (C., 1897-9.)

O.M. BARLOW, THOMAS, M.D., F.R.C.P., 10, Wimpole Street, W.

1897 BARRATT, J. O. WAKELIN, 26, Upper Wimpole Street, W.

O.M. BASTIAN, H. CHARLTON, M.D., F.R.C.P., F.R.S., 8A, Manchester Square, W. (Pres., 1892; V.P., 1891; C., 1886-9, 1893-5.)

1897 BATTEN, FREDERICK E., M.D., M.R.C.P., 124, Harley Street.

O.M. BEACH, FLETCHER, M.B., F.R.C.P., Winchester House, Kingston Hill, Surrey.

,, BEEVOR, C. E., M.D., F.R.C.P., 33, Harley St., W. (Tr., 1894-9; C. 1893.)

1888 BIRT, ERNEST, M.D., L.R.C.P., M.R.C.S., West Riding Asylum, Wakefield.

1898 BOLTON, JOSEPH SHAW, M.D., B.S., B.Sc., Mason College, Birmingham.

1896 BOND, C. HUBERT, M.D., D.Sc.Edin., London County Asylum, Bexley, Kent.

1892 BOWLBY, ANTHONY A., F.R.C.S., 24, Manchester Square, W.

1898 BOYCE, RUBERT W., M.B.Lond., University College, Liverpool.

,, BOYCOTT, ARTHUR NORMAN, M.D.Cantab., Cane Hill Asylum, Purley, Surrey.

,, BRADBURY, JOHN B., M.D.Cantab., F.R.C.P.Lond., 4, St. Peter's Terrace, Cambridge.

1889 BRADFORD, J. R., M.D., D.Sc., F.R.C.P., F.R.S., 60, Wimpole St., W.

O.M. BRAMWELL, BYROM, M.D., F.R.C.P.Ed., 23, Drumsheugh Gardens, Edinburgh. (C., 1894.)

1895 BRAMWELL, JOHN MILNE, M.B., C.M., 2, Henrietta Street, W.

1892 BRISTOWE, HUBERT CARPENTER, M.D., L.R.C.P., M.R.C.S., 18, Warwick Road, Maida Vale.

O.M. BROADBENT, Sir WM. H., Bart., M.D., F.R.C.P., F.R.S., 84, Brook Street, W. (Pres., 1895; V.P., 1893; C., 1886-7, 1896.)

Elected

1894 BRODIE, T. GREGOR, M.D., L.R.C.P., Lindfield, Uxbridge Road, Surbiton.

1897 BROWN, H. H., M.D., F.R.C.S., 22, Museum Street, Ipswich.

1888 BROWN, SANGER, M.D., 100, State Street, Chicago.

O.M. BRUCE, ALEX., M.D., F.R.C.P.Ed.. 13, Alva Street, Edinburgh. (C. 1895.)

,, BRUCE, J. MITCHELL, M.D., F.R.C.P., 23, Harley Street, W.

1898 BRUCE, SAMUEL NOBLE, M.R.C.S.Eng., 43, Kensington Gardens Square.

O.M. BRUNTON, T. LAUDER, M.D., F.R.C.P., F.R.S., 10, Stratford Place, W. (C., 1896-8.)

1895 BRYAN, FREDERICK, M.B.Dunelm, M.R.C.P., The Asylum, New Southgate, N.

,, BRYANT, JOHN HENRY, M.D.Lond., L.R.C.P., Guy's Hospital, S.E.

1890 BURY, JUDSON SYKES, M.D., F.R.C.P., 10, St. John Street, Manchester.

O.M. BUZZARD, THOMAS, M.D., F.R.C.P., 74, Grosvenor Street, W. (Pres., 1890; V.P., 1888-89; C., 1886-7, 1891-5.)

1898 CALEY, ALBERT, M.D.Lond., M.R.C.P., 24, Upper Berkeley Street, W.

1898 CAMPBELL, HARRY, M.D., B.S., F.R.C.P.Lond., 23, Wimpole St., W.

1887 CATTELL, J. MCK., M.A., Ph.D., Columbia College, New York, U.S.A.

1891 CLARKE, J. MICHELL, M.D., F.R.C.P., 28, Pembroke Road, Clifton, Bristol.

1898 CLINCH, THOMAS ALDOUS, M.D.Edin., Durham County Asylum, Winterton, Ferryhill, Co. Durham.

O.M. COBBOLD, C. S. W., M.D., F.R.C.P.Ed., Bailbrook House, Bath.

1892 COLE, ROBERT HENRY, M.D., M.R.C.P., M.R.C.S., 53, Upper Berkeley Street, Portman Square.

1890 COLMAN, W. S., M.D., F.R.C.P., 22, Wimpole Street, W. (Sec., 1899.)

,, CORNER, HARRY, M.D., L.R.C.P., Brook House, Southgate, Middlesex.

1898 CROOKSHANK, F. GRAHAM, M.D.Lond., M.R.C.S., L.R.C.P., County Asylum, Berry Wood, Northampton.

1894 CUMING, JAMES, Prof., M.S., F.R.C.P.I., 33, Wellington Place, Belfast.

1895 DAVIDSON, ANDREW, M.B., C.M., The County Asylum, Dorchester, Dorset.

1897 DAWSON, WILLIAM R., M.D., B.Ch., Farnham House, Finglas, Co. Dublin, Ireland.

1894 DEAN, H. PERCY, M.D., F.R.C.S., 69, Harley Street, W.

1898 DODD, HENRY WORK, F.R.C.S.Eng., 136, Harley Street, W.

O.M. DRESCHFIELD, JULIUS, M.D., F.R.C.P., 325, Oxford Road, Manchester. (V.P., 1898-9.)

,, DRUMMOND, DAVID, M.D., 6, Saville Place, Newcastle-on-Tyne.

1892 DUPUY, EUGENE, M.D., 53, Avenue Montaigne, Paris.

1889 ECCLES, A. SYMONS, M.B., M.R.C.S., 23, Hertford Street, Mayfair, W.

O.M. FERRIER, DAVID, M.D., F.R.C.P., F.R.S., 34, Cavendish Square, W. (Pres., 1894: V.P., 1892-3; C., 1886-90-95.)

Elected

O.M. FOSTER, MICHAEL, M.D., F.R.S., Shelford, Cambridge.

1888 FOX, ARTHUR, M.B., F.R.C.P.Ed., 16, Gay Street, Bath.

O.M. FOX, E. LONG, M.D., F.R.C.P., Church House, Clifton, Gloucestershire.

,, GASKELL, W. H., M.D., F.R.S., The Uplands, Great Shelford, Cambs. (Pres., 1899; V.P., 1897-8; C., 1896.)

,, GEE, SAMUEL, J., M.D., F.R.C.P., 31, Upper Brook Street, W.

,, GODLEE, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.

1898 GOODLIFFE, JOHN HENRY, M.D.Aberd., County Asylum, Berry Wood, Northampton.

1897 GOSSAGE, ALFRED MILNE, M.B., M.R.C.P., 54, Upper Berkeley Street.

1892 GOTCH, FRANCIS, M.A., F.R.S., The Lawn, Banbury Road, Oxford.

1893 GRANT, DUNDAS, M.D., F.R.C.S., 8, Upper Wimpole Street, W.

O.M. GUNN, R., MARCUS, M.B., F.R.C.S., 54, Queen Anne Street, W. (C., 1899.)

1895 GUTHRIE, LEONARD, M.D., M.R.C.P., 15, Upper Berkeley Street, W.

1893 HALLIBURTON, WILLIAM DOBINSON, M.D., F.R.C.P., F.R.S., 9, Ridgemount Gardens, W.C.

O.M. HAMILTON, D. J., M.B., F.R.S.E., University, Aberdeen.

1898 HANBURY, WILLIAM R., M.R.C.S., L.R.C.P., The Oaks, Longlands Road, Sidcup.

1897 HARRIS, WILFRED J., M.D., M.R.C.P., 7, Craven Mansions, Craven Terrace, W.

1898 HARTLEY, W. DARLEY, M.D. (Dunelm), M.R.C.P. (Lond.), "Mayfield," Gilmore Hill Road, Cape Town, South Africa.

,, HASLETT, JOHN HANDFIELD, M.R.C.S., L.R.C.P., Halliford House, Sunbury-on-Thames.

1891 HAWKINS, HERBERT PENNELL, M.D., F.R.C.P., 56, Portland Place, W.

O.M. HAYCRAFT, J. B., M.B., F.R.S.E., The University, Cardiff.

1891 HEAD, HENRY, M.D., M.R.C.P., 61, Wimpole Street, W.

O.M. HILL, ALEX., M.A., M.D., Downing Lodge, Cambridge. (Pres., 1896; V.P., 1895; C., 1892-94.)

,, HOPKINS, JOHN, F.R.C.S., London Sick Asylum, Cleveland Street, W.

,, HORSLEY, VICTOR, F.R.C.S., F.R.S., 25, Cavendish Square, W. (Pres., 1898; V.P., 1896-7; C., 1892-3, 1899.)

1889 HUGGARD, WILLIAM R., M.D., F.R.C.P., Davos Platz, Switzerland.

1896 HULME, GEORGE FREDERICK, M.D., M.S.Ed., Montague Road, Felixstowe.

1889 HUMPHRY, LAURENCE, M.D., M.R.C.P., 3, Trinity Street, Cambridge.

O.M. HUTCHINSON, JONATHAN, F.R.C.S., F.R.S., 15, Cavendish Square, W. (Pres., 1889; V.P., 1887-88; C., 1886, 1890.)

1889 HYSLOP, T. B., M.D., C.M., Bethlem Royal Hospital, Lambeth, S.E.

O.M. JACKSON, J. HUGHLINGS, M.D., F.R.C.P., F.R.S., 3, Manchester Square, W. (Pres., 1886; H.J.L., 1897; C., 1887-92.)

,, JESSOP, W. H., M.B., F.R.C.S., 73, Harley Street, W.

1895 JOHNSTON, GEORGE, M.D., M.R.C.P., 6, Manchester Square, W.

Elected

1892 JONES, HENRY LEWIS, M.D., F.R.C.P., 61, Wimpole Street, W.

1894 JONES, ROBERT, M.D., F.R.C.S., Claybury Asylum, Woodford, Essex.

„ KIDD, PERCY, M.D., F.R.C.P., 60, Brook Street, W.

1896 LANGDON, F. W., M.D., Cincinnati, Ohio, U.S.A.

1898 LANGDON-DOWN, REGINALD L., M.B., B.C.Cantab., M.R.C.P.Lond., 81, Harley Street, W.

O.M. LANGLEY, J. N., M.A., D.Sc., F.R.S., Trinity College, Cambridge (C., 1898-9.)

1888 LAWFORD, J. B., M.D., F.R.C.S., 99, Harley Street, W.

O.M. LEES, D. B., M.D., F.R.C.P., 22, Weymouth Street, W.

1897 LEWIS, H. WOLSELEY, M.R.C.S., L.R.C.P., Claybury Asylum, Woodford, Essex.

„ LOWENTHAL, MAX, M.D., M.R.C.P., 84, Princes' Road, Liverpool.

1898 MACCORMAC, JOHN M'GEE, M.D.Durh., L.R.C.P., L.R.C.S.Edin., 31, Victoria Place, Belfast.

„ MCALDOWIE, ALEXANDER M., M.D.Aberd., 6, Brook Street, Stoke-on-Trent.

1891 MACDONALD, PETER WILLIAM, M.D., C.M., The County Asylum, Dorchester, Dorset.

„ MACKENZIE, HECTOR WILLIAM GAVIN, M.D., F.R.C.P., 59, Welbeck Street, W.

O.M. MACKENZIE, STEPHEN, M.D., F.R.C.P., 18, Cavendish Square, W. (C., 1896-8.)

1896 MACKINTOSH, ASHLEY W., M.A., M.D., Wellington House, Alford Place, Aberdeen.

1889 MACPHAIL, S. R., M.D., Borough Asylum, Rowditch, Derby.

O.M. MAGUIRE, ROBERT, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93; C., 1893-96.)

„ MANN, J. DIXON, M.D., F.R.C.P., 16, St. John's Street, Manchester.

1894 MARTIN, SIDNEY, M.D., F.R.C.P., F.R.S., 10, Mansfield Street, W.

1893 MAUDE, ARTHUR, L.R.C.P., M.R.C.S., Westerham, Kent.

1891 MAY, W. PAGE, M.D., M.R.C.P., Helouan, Cairo, Egypt.

1890 MENZIES, WILLIAM FRANCIS, M.D., B.Sc., M.R.C.P., County Asylum, Rainhill, Lancs.

O.M. MERCIER, C., M.B., M.R.C.P., Flower House, Southend, Catford, S.E. (C. 1899.)

1897 MEYER, ADOLF, M.D., Clark University, Worcester, Mass., U.S.A.

O.M. MICKLE, W. JULIUS, M.D., F.R.C.P., Grove Hall Asylum, Bow, E. (C., 1895-97.)

1895 MOORE, NORMAN, M.D., F.R.C.P., 94, Gloucester Place, W.

O.M. MORRIS, MALCOLM, F.R.C.S.Ed., 8, Harley Street, W.

1888 MOTT, F. W., M.D., F.R.C.P., F.R.S., 25, Nottingham Place, W. (Sec., 1894-8; C., 1899.)

1894 MURRAY, GEORGE R., M.A., M.D., F.R.C.P., 11, Ellison Place, Newcastle-on-Tyne.

Elect

1889 MURRAY, H. MONTAGUE, M.D., F.R.C.P., 27, Saville Row, W.

O.M. NETTLESHIP, EDWARD, F.R.C.S., 5, Wimpole Street, W.

,, NICOLSON, DAVID, C.B., M.D., M.R.C.P.Ed., Elmhyrst, Guildford, Surrey.

1888 NIERMEYER, J. H. H., M.D., Amsterdam.

,, NORMAN, CONOLLY, F.R.C.P.I., F.R.C.S.I., Richmond Asylum, Dublin.

1887 OGILVIE, GEORGE, M.B., M.R.C.P., 22, Welbeck Street, W.

,, OGILVIE, LESLIE, M.B., M.R.C.P., 46, Welbeck Street, W.

,, OLIVER, THOMAS, M.D., F.R.C.P., 7, Ellison Place, Newcastle-on-Tyne.

O.M. ORANGE, WM., C.B., M.D., F.R.C.P., The Bryn, Godalming, Surrey.

,, ORD, W. M., M.D., F.R.C.P., 37, Upper Brook Street, W. (C., 1893-94.

,, ORMEROD, J. A., M.D., F.R.C.P., 25, Upper Wimpole Street, W. (C., 1895-7.)

,, PAGE, HERBERT WILLIAM, M.A., F.R.C.S., 146, Harley Street, W. (C., 1891-2.)

1895 PARKINSON, JOHN PORTER, M.D.Lond., M.R.C.P., 40, Wimpole Street, W.

1896 PATERSON, DONALD ROSE, M.D.Edin., M.R.C.P., 18, Windsor Place, Cardiff.

1895 PINDER, GEORGE, M.B., B.C., Seafield House, Ramsey, Isle of Man.

1888 PITT, G. N., M.D., F.R.C.P., 15, Portland Place, W.

O.M. POORE, G. VIVIAN, M.D., F.R.C.P., 32, Wimpole Street, W. (C., 1898-9.)

O.M. PURVES, W. LAIDLAW, M.D., 20, Stratford Place, W.

1894 RANSOM, WILLIAM B., M.D., F.R.C.P., The Pavement, Nottingham.

1890 RAYNER, HENRY, M.D., M.R.C.P.Ed., 2, Harley Street, W.

O.M. REID, E. W., M.B., University College, Dundee.

1897 REISSMANN, CHARLES H., B.A., B.Sc., Saxony Villas, Oppidans Road, Primrose Hill.

1893 RENNIE, GEORGE E., M.D.

1895 REYNOLDS, ERNEST S., M.D., F.R.C.P., 23, St. John Street, Manchester.

1892 RICHARDS, JOSEPH PEEKE, M.R.C.S., 6, Freeland Road, Ealing, W.

1891 RIVERS, W. H. R., M.A., M.D., M.R.C.P., St. John's College, Cambridge. (C. 1897-8.)

1898 ROBERTSON, WILLIAM FORD, M.D., C.M.Edin., 12, Bristo Place, Edinburgh.

1892 ROWE, EDMUND LEWIS, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.

,, RUFFER, MARC ARMAND, M.D., Cairo.

1889 RUSSELL, J. S. RISIEN, M.D., F.R.C.P., 4, Queen Anne Street, W. (Sec., 1896-9.)

O.M. SANDERSON, J. BURDON, M.D., F.R.C.P., F.R.S., Banbury Road, Oxford.

,, SAVAGE, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square, (Pres., 1897; V.P., 1895; C., 1886-90, 1898.)

Elected

1888 SAVILL, T. D., M.D., M.R.C.P., 60, Upper Berkeley Street, W.

O.M. SCHÄFER, E. A., F.R.S., University College, Gower Street. (Pres., 1893; V.P., 1892; C., 1886-90.)

,, SEMON, Sir FELIX, M.D., F.R.C.P., 39, Wimpole Street, W. (C., 1899.)

1898 SEWARD, WM. JOSEPH, M.B.Lond., Colney Hatch Asylum, N.

O.M. SHARKEY, S. J., M.D., F.R.C.P., 22, Harley Street, W. (C., 1889-92.)

,, SHERRINGTON, C. S., M.D., F.R.S., 16, Grove Park, Liverpool. (C. 1892-95.)

1894 SHUTTLEWORTH, GEORGE E., M.D., Ancaster House, Richmond.

1898 SIMPSON, FRANCIS O., L.R.C.P.Lond., M.R.C.S.Eng., Hawkhead Asylum, Paisley, Scotland.

1895 SMITH, F. J., M.D., M.R.C.P., 4, Christopher Street, Finsbury Square, E.C.

1887 SMITH, R. PERCY, M.D., F.R.C.P., 36, Queen Anne Street, W.

1895 SMITH, TELFORD, M.D., B.S., Royal Albert Asylum, Lancaster.

,, SPICER, WILLIAM T. H., M.D.Cantab., F.R.C.S., 47, Welbeck Street, W.

,, STANSFIELD, THOS. E. K., M.B., Claybury Asylum, Woodford, Essex.

,, STANLEY, DOUGLAS, M.D.Edin., 9, Easy Row, Birmingham.

1892 STARLING, ERNEST HENRY, M.D., F.R.C.P., B.S., 8, Park Square, N.W.

1897 STEWART, PURVES, M.A., M.D., M.R.C.P., 133, Harley Street, W.

O.M. STEWART, Sir T. GRAINGER, M.D., F.R.C.P.Ed., 19, Charlotte Square, Edinburgh.

,, STIRLING, WM., M.D., D.Sc., Owens College, Manchester.

1897 STODDART, WM. H., M.D., B.S., M.R.C.P.Lond., Bethlem Royal Hospital, Lambeth, S.E.

1895 STOUT, G. F., M.A., St. John's College, Cambridge.

1887 SUCKLING, C. W., M.D., M.R.C.P., 103, Newhall Street, Birmingham.

O.M. SULLY, JAMES, M.A., LL.D., 1, Portland Villas, Hampstead. (V.P., 1890-91.)

1889 SUTHERLAND, HENRY, M.D., M.R.C.P., 21, New Cavendish Street, Portland Place, W.

1888 SYERS, HENRY W., M.D., M.R.C.P., 4, Oxford and Cambridge Mansions, Hyde Park, W.

1898 TAYLOR, F. R. P., M.D., B.S.Lond., Darenth Asylums and Schools, Dartford, Kent.

1891 TAYLOR, JAMES, M.D., F.R.C.P., 49, Welbeck Street, W.

1898 THOMSON, H. CAMPBELL, M.D., M.R.C.P., 34, Queen Anne Street, W.

1889 THORBURN, WM., F.R.C.S., The Limes, Nelson Street, Manchester.

1892 TITCHENER, E. B., B.A., Ph.D., 72, Heustis Street, Ithaca, N.Y.

O.M. TOOTH, H. H., M.D., F.R.C.P., 34, Harley Street, W. (Sec., 1891-95; C., 1896-9.)

1892 TREVELYAN, E. F., M.D., B.Sc., M.R.C.P., 40, Park Square, Leeds.

1892 TUCKEY, CHARLES LLOYD, M.D., C.M., 88, Park Street, Grosvenor Square, W.

1892 TUKE, Sir J. BATTY, M.D., F.R.C.P.Ed., 20, Charlotte Sq., Edinburgh.

Elected

1891 TUKE, THOMAS SEYMOUR, M.B., M.R.C.S., Chiswick House, Chiswick.

,, TURNER, WILLIAM ALDREN, M.D., F.R.C.P., 13, Queen Anne Street, W.

1898 TURNEY, HORACE G., M.B., M.Ch.Oxon, F.R.C.P.Lond., 68, Portland Place, W.

O.M. TWEEDY, JOHN, F.R.C.S., 100, Harley Street, W.

1888 VOORTHUS, J. A., M.D., Apeldoorn, 542, Loulaan, Netherlands.

1895 WADE, ARTHUR L., M.D., Somerset and Bath Asylum, Wells.

1894 WALKER, A. STODART, M.B., 30, Walker Street, Edinburgh.

O.M. WALLER, AUGUSTUS, M.D., F.R.S., 16, Grove End Road, N.W. (V.P., 1899; C., 1894-7.)

O.M. WARD, J., D.Sc., Trinity College, Cambridge.

,, WARNER, FRANCIS, M.D., F.R.C.P., 5, Prince of Wales Terrace, Kensington Palace, W.

1892 WASHBOURN, J. W., M.D., F.R.C.P., 6, Cavendish Place, W.

O.M. WATTEVILLE, A. DE, M.A., M.D., B.Sc., 30, Welbeck Street, W. (C., 1890-95, 1896-9; Sec., 1886-9; Editor of *Brain*.)

1897 WEBER, F. PARKES, M.D., F.R.C.P., 19, Harley Street, W.

,, WEST, SAMUEL, M.D.. F.R.C.P., 15, Wimpole Street, W.

,, WHITE, ERNEST W., M.B., M.R.C.P., City of London Asylum, Dartford.

O.M. WHITE, W. HALE, M.D., F.R.C.P., 65, Harley Street, W. (C., 1897-9.)

1894 WHITING, ARTHUR J., M.D., M.R.C.P., 142, Harley Street, W.

1889 WIGLESWORTH, JOSEPH, M.D., F.R.C.P., County Asylum, Rainhill, Lancashire.

1894 WILLIAMSON, RICHARD T., M.D., M.R.C.P., 294, Oxford Road, Manchester.

1896 WILSON, ALBERT, M.D.Edin., M.R.C.P., 33, Fairlop Road, Leytonstone.

,, WOOD, GUY M., M.B., M.R.C.P., County Asylum, Rainhill, Lancashire.

1889 WOOD, T. OUTTERSON, M.D., M.R.C.P., 40, Margaret Street, Cavendish Square, W.

1892 WOODHEAD, G. SIMS, M.D., F.R.C.P.Ed., Examination Hall, Victoria Embankment.

1898 YOUNG, ROBERT ARTHUR, M.D., B.Sc., M.R.C.P., 41, Wimpole St., W.